# ANALYSIS OF CLINICAL PROFILE, CARDIAC ARRHYTHMIAS AND ELECTROLYTE DISTURBANCES IN PATIENTS WITH ACUTE YELLOW OLEANDER POISONING

***Dissertation submitted in partial fulfillment of requirements for***

M.D. DEGREE IN GENERAL MEDICINE

BRANCH I

Of

THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY,
CHENNAI, INDIA.

MADRAS MEDICAL COLLEGE,
CHENNAI 600003
MARCH 2010

# ACKNOWLEDGEMENT

AT THE OUTSET, I THANK **PROF.J. MOHANASUNDARAM M.D.,** DEAN, MADRAS MEDICAL COLLEGE AND GOVERNMENT GENERAL HOSPITAL, CHENNAI-3 FOR HAVING PERMITTED ME TO USE HOSPITAL DATA FOR THE STUDY.

I AM GRATEFUL TO **PROF. C. RAJENDIRAN, M.D.,** DIRECTOR AND HEAD OF DEPARTMENT, INSTITUTE OF INTERNAL MEDICINE, MADRAS MEDICAL COLLEGE AND GOVERNMENT GENERAL HOSPITAL, CHENNAI-3 FOR HIS SUPPORT.

I WOULD ALSO LIKE TO THANK ASSISTANT PROFESSORS **DR.R.MUTHUSELVAN, M.D.,** AND **DR.S.BASKAR, M.D.,** MADRAS MEDICAL COLLEGE AND GOVERNMENT GENERAL HOSPITAL, CHENNAI-3 FOR THEIR SUPPORT AND GUIDANCE.

I ALSO EXPRESS MY GRATITUDE TO **DR.D.THANGAM,** ASSISTANT PROFESSOR, TOXICOLOGY, MADRAS MEDICAL COLLEGE AND GOVERNMENT GENERAL HOSPITAL, CHENNAI-3.

MY SINCERE WISHES TO ALL THE PATIENTS WHO PARTICIPATE IN THIS STUDY.

LASTLY, I THANK ALL MY PROFESSIONAL COLLEAGUES FOR THEIR SUPPORT, VALUABLE CRITICISMS AND SUGGESSTION.

# CERTIFICATE

This is to certify that the dissertation entitled "**ANALYSIS OF CLINICAL PROFILE, CARDIAC ARRHYTHMIAS AND ELECTROLYTE DISTURBANCES IN PATIENTS WITH ACUTE YELLOW OLEANDER POISONING**" is a bonafide work done by **Dr. G.indhumathi**, at Madras Medical College, Chennai in partial fulfillment of the university rules and regulations for award of M.D., Degree in General Medicine (Branch-I) under my guidance and supervision during the academic year 2007 -2010.

**Prof. C. RAJENDIRAN, M.D.,**
**Director and Professor,**
**Institute of Internal Medicine**
**Madras Medical College &**
**Govt. General Hospital,**
**Chennai -3**

**Prof.J.MOHANASUNDARAM, M.D.,**
**THE DEAN**
**Madras Medical College &**
**Govt. General Hospital,**
**Chennai – 600 003**

# CONTENTS

# INTRODUCTION

The yellow oleander (Thevetia Peruviana) is an ornamental tree, which is common throughout the tropics and subtropics. It contains cardiac glycosides that are toxic to cardiac muscle and the autonomic nervous system. Deliberate ingestion of oleander seeds has become a popular method of self-harm in India.

Ingestion of oleander seeds results in a clinical picture similar to that of digoxin overdose. Severely poisoned patients may die in dc shock resistant ventricular fibrillation. Severe Hyperkalemia is a feature of acute oleander poisoning, which may further worsen toxicity and lead to serious Arrhythmias.

So, it was decided to study the yellow oleander poisoning in our place regarding the clinical profile, ECG changes and Electrolytes changes which decides the mortality and modification of which would improve the prognosis in our patients.

## REVIEW OF LITERATURE

Oleander is an ornamental tree of the Apocyanaceae family that is common throughout the tropics and subtropics.[1] It is wide spread in India, Nepal and Srilanka. Its flowers are used as offerings in the temple. Its sap contains cardiac glycosides (thevetins A and B and neriifolin) including the roots and the smoke produced from burning, toxic to cardiac muscle and the ingestion of its seeds results in a clinical picture similar to that of digoxin overdose.[3-5] . Oleander leaf also contains other biologically active constituents that have antimitotic and insecticidal properties. Oleander is also reported to have emetogenic, cathartic, insecticidic, parasiticidic, anthelmintic, menstrual stimulant, and abortifacient activities[30].

The majority of deaths occurring after ingestion of plant are due to yellow Oleander or 'pila kaner' (cerebra thevetia), pink eyed cerebra or 'sea mango' (cerebra manghas), and white oleander or 'kaner' (nerium odorum) are reported in South India. Poisoning with another related plant cerebra odollum is a common occurrence in Kerala with as many as 50% of the plant poisoning caused by this plant[14].

Adults have died after consuming oleander leaves in herbal teas.[8] Accidental poisonings occur throughout the tropics particularly in children[2 6 7]. Many patients with moderate poisoning show PR interval prolongation and progression to atrioventricular (AV) dissociation. Severely poisoned patients may die due to dc shock resistant ventricular fibrillation. However, deliberate ingestion of yellow oleander seeds has recently become a popular method of self harm [9 10].

## PATHOPHYSIOLOGY

More than 200 naturally occurring cardiac glycosides have been identified. The seed contains four percent of the thevetin, which is one-eighth as potent as ouabin and similar to digitalis in action; thevetoxin is similar to but less toxic than thevetin; nerifolin(more potent than thevetin); peruvoside, and ruvoside, cerberin and also bitter principle that acts on the CNS, and produce tetanoid convulsions.[11]


**Fig 1: Molecular structure of oleanderin.**

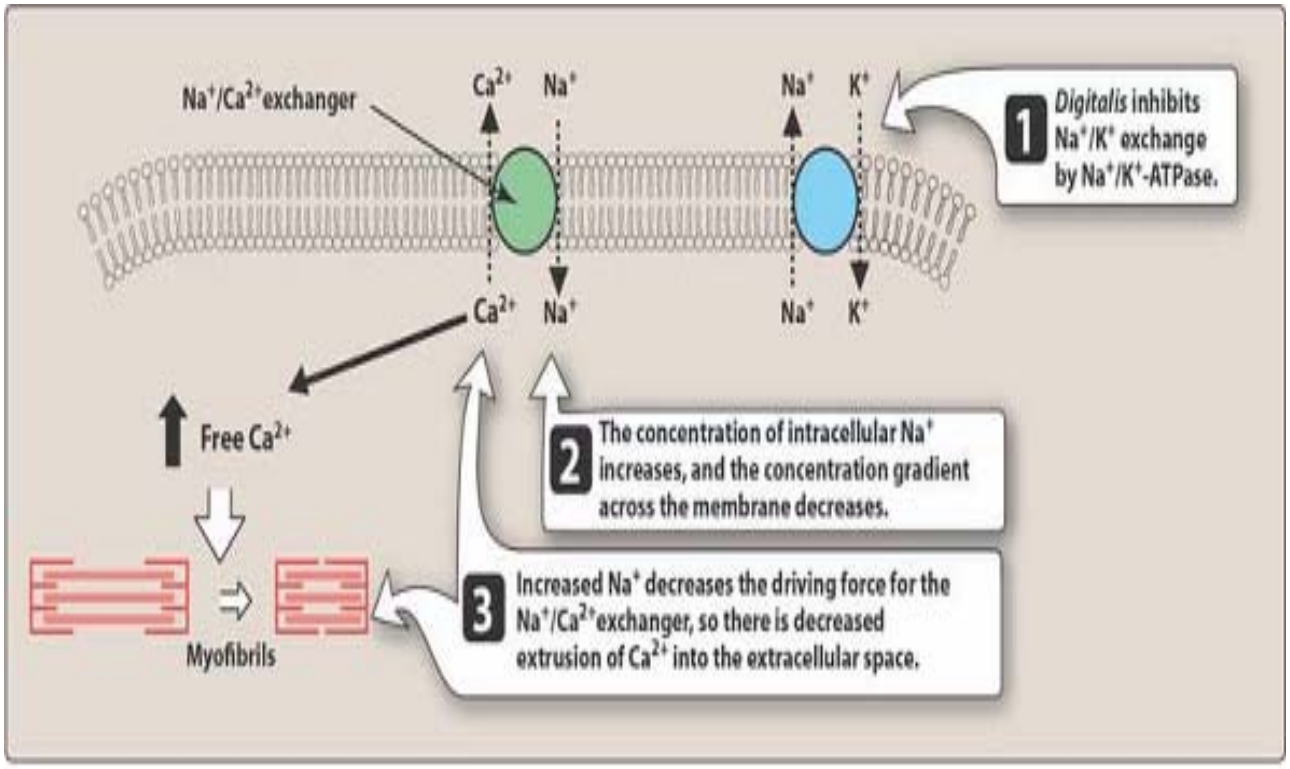



**Fig 2: Mechanism of action of cardiac glycosides (ATPase = adenosine triphosphatase).**

These bind to a site on the cell membrane, producing reversible inhibition of the sodium ($Na^+$)-potassium ($K^+$)-adenosine triphosphatase (ATPase) pump, which causes increased intracellular sodium and decreased intracellular potassium. In myocytes, elevated intracellular sodium concentrations produce increased intracellular calcium concentrations via a $Na^+$ -calcium ($Ca^{++}$)-exchanger. In response to the increased intracellular calcium, the sarcoplasmic reticulum releases additional calcium intracellularly resulting in depolarization of the cell[l2].

Cardiac glycosides also have vagotonic effects, resulting in bradycardia and heart blocks.

The pathophysiology that produces cardio toxicity involves prolonging refractory period in atrioventricular (AV) node, shortening refractory periods in atria and ventricles, and decreasing resting membrane potential (increased excitability). At therapeutic doses, cardiac glycosides also may increase inotropy. Any dysrhythmia characterized by both increased automaticity and depressed conduction is suggestive of cardiac glycoside toxicity.

Inhibition of $Na^+$ -$K^+$ -ATPase in skeletal muscle results in increased extra cellular potassium and contributes to hyperkalemic state, which may lead to arrhythmias and death[13].

**Lethal dose**

Previously suggested that the lethal dose was between four and seven seeds.

But, even the ingestion of 1–2 seeds by a young child is more dangerous, since small amounts of seeds may not always induce vomiting.

Recent studies show that there is no simple relationship between the number of seeds ingested and outcome.

So we have to monitor the degree of poisoning by following the cardiac rhythm rather than infer from the number of seeds taken[28-29].

**CLINICAL FEATURES**

The symptoms start within 2-3 hours of ingestion[11]. Oleander may cause irritation to the mucosal membranes, resulting in burning sensation around the mouth and increased salivation.

The cardiac glycosides in oleander produce more gastrointestinal effects than those in digoxin, and the symptoms range from nausea and vomiting to cramping and bloody diarrhea[39].

Confusion, dizziness, drowsiness, weakness, visual disturbances, mydriasis and convulsions are central nervous system manifestations of toxicity[2-6].

The most serious side effects of oleander poisoning are cardiac. Bradycardia and heart block are the most frequently reported cardiac

abnormalities. Various ventricular dysrhythmias and tachyarrhythmias have been described.[3-5, 7-9].

The time course and outcome was also quite variable. It also depends upon the form of the seeds they consumed. In SriLanka peoples usually eat the seeds as a whole, which probably reduces the bioavailability of the cardiac glycosides.

But in south India peoples crush the seeds and drink, from which the cardiac glycosides might be more quickly absorbed.

Patients may be in Sinus Rhythm for quite long period before developing serious Arrhythmias. So it's important to monitor all the patients at least for 48 hrs[30].

**ECG CHANGES**

The most common arrhythmias were conduction defects affecting Sinus node

- Sinus bradycardia - 25%
- Sinus arrest or exit block - 62%

AV node

- First, Second and third degree heart block - 53%.

Both sinus and AV nodes - 30%[16.]

Mobitz type II AV conduction block occurred in the oleander patients, but rare in isolated digoxin poisoning[24,40].

Atrial fibrillation and flutter were uncommon in oleander patients, probably because of their good pre-existing cardiac status. Patients with these arrhythmias presented with a slow ventricular response owing to impaired AV conduction, as occurs in digoxin poisoning.

Ventricular ectopic beats were uncommon in the oleander patients.

**ELECTROLYTES**

Hypokalemia worsens toxicity due to digitalis glycosides, and hyperkalemia is life threatening. Both must be corrected. Hyperkalemia is due to extra cellular shift of potassium rather than an increase in total body potassium, and is a marker of a poor outcome in cardiac glycoside poisoning.

Even though all symptomatic patients had persistent vomiting, severe yellow oleander induced cardio toxicity was associated with hyperkalaemia. The degree of hyperkalaemia correlated with the serum digoxin cross reactive cardiac glycoside concentration[25].

Intravenous calcium increases the risk of cardiac arrhythmias and is not recommended in treating hyperkalemia. Oral or rectal administration of sodium polystyrene sulfonate resin may result in hypokalemia when used together with digoxin-specific antibody fragments.

Unlike digoxin toxicity, serum magnesium concentrations are less likely to be affected in yellow oleander poisoning. The effect of magnesium concentrations on toxicity and outcome is not known. Hypomagnesaemia should be corrected as it can worsen cardiac glycoside toxicity.

**SERUM CARDIAC - GLYCOSIDES**

The cross-reactivity seen between digoxin radioimmunoassays and the glycosides of oleander is well known. This cross-reactivity can at least identify the presence of a cardio active glycoside in the case of an unknown poisoning.

Beyond its qualitative usefulness in oleander toxicity, the digoxin serum level's clinical significance is unknown. The digoxin radioimmunoassay is not specific for all of the glycosides that may be present in the oleander plant[16-23].

The use of a digoxin radioimmunoassay in oleander toxicity will only indicate the presence of glycoside and may not indicate the degree of toxicity.

There was no apparent relation between the site of conduction block and the serum cardiac glycoside or electrolyte values. However, the mean

Serum cardiac glycoside concentration was significantly higher in patients with both AV and sinus node block than in patients with block affecting only the sinus node[25].

## TREATMENT

### PREHOSPITAL CARE

- Prehospital care should focus on ABCs, with special emphasis on supporting respiratory and cardiac function.
- During transport, the patient should receive supplemental oxygen and an IV line. Cardiac and pulse oximeter monitoring should be continuous.
- In patients with protected airway and normal mental status, activated charcoal can be administered.
- Atropine should be given to patients with clinically significant bradycardia (e.g., hypotension, change of mental status).

### EMERGENCY DEPARTMENT CARE

The treatment of oleander poisoning is empirically based on the treatment of digitalis-glycoside toxicity and consists of supporting the patient hemodynamically.

No definite criteria are available for risk stratification

### PREVENT FURTHER ABSORPTION

### STOMACH WASH AND MULTI-DOSE ACTIVATED CHARCOAL

Activated charcoal is most effective when used soon after ingestion of the toxin.

It interrupts the enterohepatic circulation of the glycoside. But, not all the glycosides have enterohepatic circulation.

Activated charcoal prevents further absorption of the cardiac glycosides[17]. So, it increases the excretion of digoxin in humans.[27,28]

Activated charcoal improves the outcome in oleander poisoning and reduces the need for expensive cardiac pacing and antidigoxin antibody Fab fragments.

So administering activated charcoal is beneficial without any risk.

Dosage - 50 g of activated charcoal every 6 h for 3 days.

**MONITORING OF SERUM LEVELS OF POTASSIUM AND TREATMENT OF FLUID, ELECTROLYTE AND ACID-BASE BALANCE**

**HYPERKALEMIA:**

Life-threatening hyperkalemia (>6.5 mEq/L) may be seen with acute toxicity and results from a redistribution phenomenon rather than increased body stores.

- Glucose, insulin, sodium bicarbonate, and albuterol may be used to facilitate redistribution of potassium intracellularly. However, albuterol may precipitate cardiac dysrhythmias.
- Calcium should be avoided. A recent study shows that, in contrast to earlier studies, IV calcium administration to treat hyperkalemia

secondary to cardiac glycoside toxicity resulted in no benefit or harm. However, it's not recommended.

- Life-threatening hyperkalemia should be treated with Fab fragments.

## HYPOKALEMIA

Symptomatic or severe hypokalemia should be corrected with intravenous potassium preparations.

Potassium supplements:

Potassium chloride (also citrate, acetate, bicarbonate, gluconate).

Potassium chloride is the preferred salt for patients with preexisting alkalosis and first choice for IV therapy.

**Adult Dose:**

IV replacement:

10-40 mEq IV infused over 2-3 h; infusion rate not to exceed 40 mEq/h; may be repeated q3-4h; Please modify infusion rate for specific requirements.

PO supplementation:

50-100 mEq/d PO divided bid/tid or qd as SR formulation; larger doses may be needed in severe depletion to replenish potassium body storage.

**Precautions**

Do not infuse rapidly; high plasma concentrations of potassium may cause death due to cardiac depression, arrhythmias, or arrest;

Plasma levels do not necessarily reflect tissue levels; monitor potassium replacement therapy whenever possible by means of continuous or serial ECG;

IV potassium must be diluted before administration, when a concentration >40 mEq/L is infused, local pain and phlebitis also may follow**.**

## MONITORING THE CARDIAC RHYTHM.

This treatment may include,

- Bradydysrhythmias: Administering atropine for severe bradycardia. If atropine is not rapidly successful, consider use of isoproterenol, administration of Fab fragments and transcutaneous cardiac pacing.
- Tachydysrhythmias: Phenytoin and lidocaine may be used as antidysrhythmics if Fab fragments are not immediately

available (which decrease automaticity without slowing AV nodal conduction and increase fibrillation threshold) may be used to treat ventricular dysrhythmias.

- Use cardioversion only as a last resort. It should be attempted after a loading dose of phenytoin and at a significantly reduced initial power setting of 5-10 J.
- Quinidine and procainamide may enhance cardiac glycoside toxicity by slowing conduction across AV node; both should be avoided.
- Beta-blockers and calcium channel blockers have questionable value[14, 26].

**.ADMINISTER ANTIDOTE: Digoxin-specific Fab antibody fragments (Digibind)**

Sheep-derived digoxin antibody Fab fragments reportedly are effective for some plant cardiac glycosides. Digibind binds with an unknown portion of the total glycosides. Indications for digoxin antibody Fab fragments are the same for both pharmaceutical as well as nonpharmaceutical cardiac glycoside toxicity and include the following:

- Hyperkalemia (>5.0 mEq/L) in acute toxicity
- Life-threatening supraventricular and ventricular dysrhythmias

- Hemodynamically significant bradycardia unresponsive to atropine
- Chronic digoxin toxicity with dysrhythmias, significant GI symptoms, acute altered mental status, or renal insufficiency
- Serum digoxin level >15 mg/mL at any time
- Poisoning by nondigoxin cardiac glycoside
- To aid in treatment of suspected cardiac glycoside poisoning without a confirmatory level

Forced diuresis, hemoperfusion, and hemodialysis are ineffective in enhancing the elimination.

**INDICATIONS OF PACE MAKERS IN BRADY-ARRHYTHMIAS**

| **Table 1 Summary of Guidelines for Pacemaker Implantation in SA Node Dysfunction** |
| --- |
| Class I |
| 1. SA node dysfunction with symptomatic bradycardia or sinus pauses |
| 2. Symptomatic SA node dysfunction as a result of essential long-term drug therapy with no acceptable alternatives |
| 3. Symptomatic chronotropic incompetence |
| Class IIa |
| 1. SA node dysfunction with heart rates < 40 beats/min without a clear and consistent relationship between bradycardia and symptoms |
| 2. SA node dysfunction with heart rates < 40 beats/min on an essential long-term drug therapy with no acceptable alternatives, without a clear and consistent relationship between bradycardia and symptoms |
| 3. Syncope of unknown origin when major abnormalities of SA node dysfunction are discovered or provoked by electrophysiologic testing |
| Class IIb |

| |
|---|
| 1. Mildly symptomatic patients with waking chronic heart rates < 40 beats/min |
| Class III |
| 1. SA node dysfunction in asymptomatic patients even those with heart rates < 40 beats/min |
| 2. SA node dysfunction in which symptoms suggestive of bradycardia are not associated with a slow heart rate |
| 3. SA node dysfunction with symptomatic bradycardia due to nonessential drug therapy |

**Table 2 Guideline Summary for Pacemaker Implantation in Acquired AV Block**

| |
|---|
| Class I |
| 1. Third-degree or high-grade AV block at any anatomic level associated with: |
| a. Symptomatic bradycardia |
| b. Essential drug therapy that produces symptomatic bradycardia |
| c. Periods of asystole > 3 s or any escape rate < 40 beats/min while awake |
| d. Postoperative AV block not expected to resolve |

| |
|---|
| e. Catheter ablation of the AV junction |
| f. Neuromuscular diseases such as myotonic dystrophy, Kearns-Sayre syndrome, Erb dystrophy, and peroneal muscular atrophy, regardless of the presence of symptoms |
| 2. Second-degree AV block with symptomatic bradycardia |
| 3. Type II second-degree AV block with a wide QRS complex with or without symptoms |
| Class IIa |
| 1. Asymptomatic third-degree AV block regardless of level |
| 2. Asymptomatic type II second-degree AV block with a narrow QRS complex |
| 3. Asymptomatic type II second-degree AV block with block within or below the His at electrophysiologic study |
| 4. First- or second-degree AV block with symptoms similar to pacemaker syndrome |
| Class IIb |
| 1. Marked first-degree AV block (PR interval > 300 ms) in patients with LV dysfunction in whom shortening the AV delay would improve hemodynamics |

| |
|---|
| 2. Neuromuscular diseases, such as myotonic dystrophy, Kearns-Sayre syndrome, Erb dystrophy, and peroneal muscular atrophy, with any degree of AV block regardless of the presence of symptoms |
| Class III |
| 1. Asymptomatic first-degree AV block |
| 2. Asymptomatic type I second-degree AV block at the AV node level |
| 3. AV block that is expected to resolve or is unlikely to recur (Lyme disease, drug toxicity) [31] |

## OBJECTIVES

To analyze the patients with acute yellow Oleander seed Poisoning with reference to-

- Socio-demographic aspects
- Clinical Profile
- ECG Changes
- Electrolyte Status and
- Outcome.

## MATERIALS & METHODS

**Setting:** Poison Control, Training and research centre Govt.General Hospital & Madras Medical College, Chennai.

**Design of Study**: Descriptive Study.

**Duration**: Jan 2009 to June 2009.

**Ethical Clearance:** Obtained from appropriate authorities.

**Informed Contest:** Obtained.

### DATA COLLECTION

Clinical parameters, CBC, Blood sugars, Serum Urea, Creatinine, Serum Na+, Serum K+, LFT, ABG, ECG, and Gastric aspirate analysis.

### BRIEF PROCEDURE:

All patients admitted with consumption of yellow oleander seed or its extracts within 48 hours were studied. The study was carried out over a six-month-period beginning Jan 2009 to June 2009..A detailed history was obtained and patients were subjected to thorough clinical examination as well as ECG soon after admission. They were assessed on a hourly basis for the

first 6 hours, 12 th hourly for next 72 hours on then daily basis until complete recovery. Results of biochemical investigations including serum electrolytes will be entered in a pre-designed proforma. Serial ECGs was collected.

**Inclusion criteria:**

Patients admitted to the toxicology ward within 48 hours of ingestion of poison.

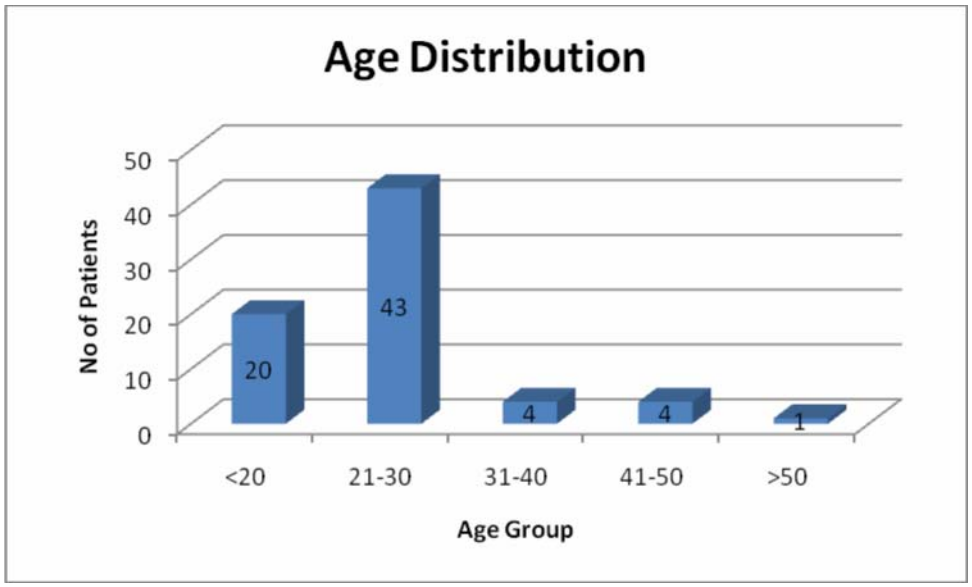
**Exclusion criteria:**

1. Patients with history of cardiovascular disease
2. Patients on cardiac drugs.
3. Patients with chronic renal failure.
4. Patients on diuretics.

## RESULTS

Epidemiological pattern of Oleander Seed Poisoning in poison centre, Govt.General Hospital

**Fig: 3**




## Table : 3

| SI NO | Age Group | Frequency(n) | Percentage(%) |
|---|---|---|---|
| 1 | <20 | 20 | 27.77 |
| 2 | 21-30 | 43 | 59.72 |
| 3 | 31-40 | 4 | 5.55 |
| 4 | 41-50 | 4 | 5.55 |
| 5 | >50 | 1 | 1.38 |

Majority of the patients were in 21-30 age groups. The number of people between the age group of 14-30 accounts for 85%.

**Fig : 4**

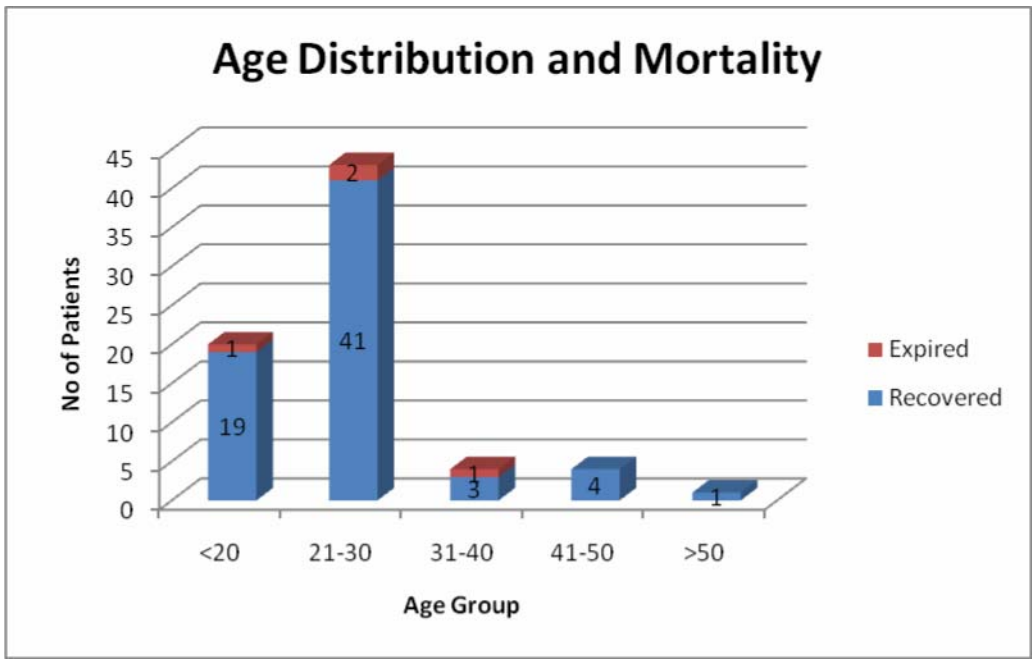



**Table : 4**

| SI No | Age Group | Frequency(n) | Death | Percentage(%) |
|---|---|---|---|---|
| 1 | <20 | 20 | 1 | 1.4 |
| 2 | 21-30 | 43 | 2 | 2.7 |
| 3 | 31-40 | 4 | 1 | 1.4 |
| 4 | 41-50 | 4 | 0 | 0 |
| 5 | >50 | 1 | 0 | 0 |

Three people in the age group of below thirty died.

**Fig: 5**

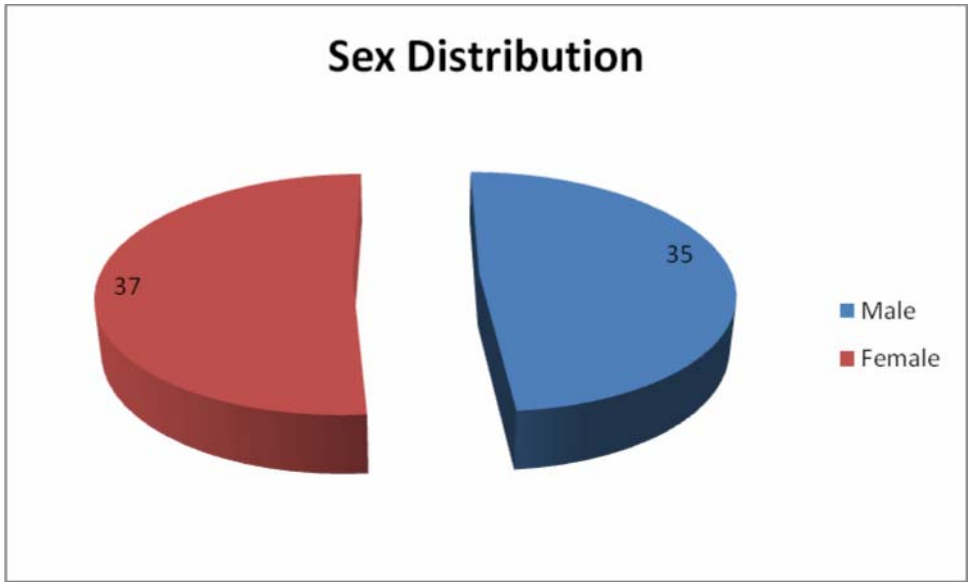



**Table : 5**

| SI NO | Sex | Frequency(n) | Percentage (%) |
|---|---|---|---|
| 1 | Male | 35 | 48.6 |
| 2 | Female | 37 | 51.4 |

Incidence of Oleander was almost equal in both genders

**Fig: 6**

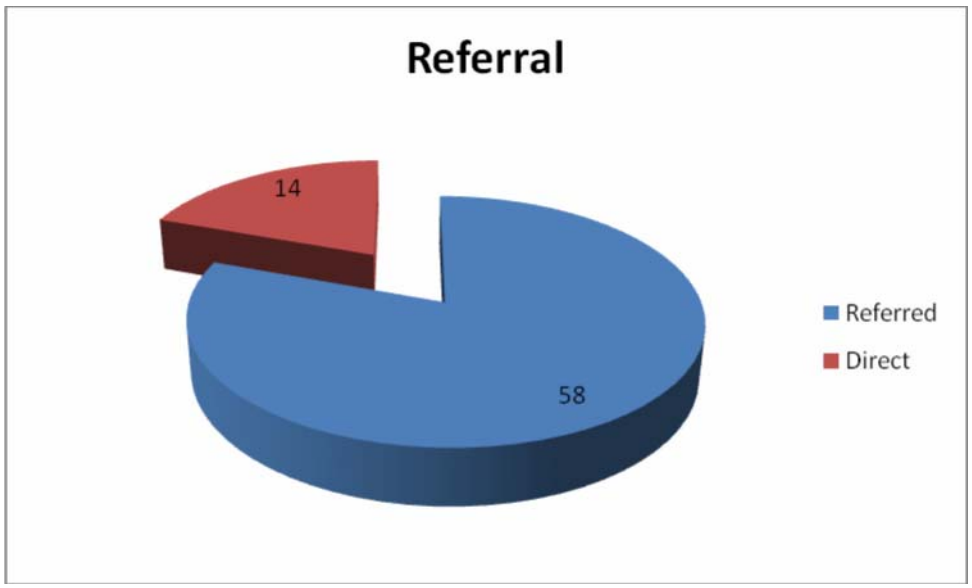



**Table: 6**

| SI No | Referral | Frequency(n) | Percentage (%) |
|---|---|---|---|
| 1 | Referred | 58 | 80.5 |
| 2 | Direct | 14 | 19.5 |

More than 80% of the patients with Oleander poisoning were referred.

**Fig: 7**

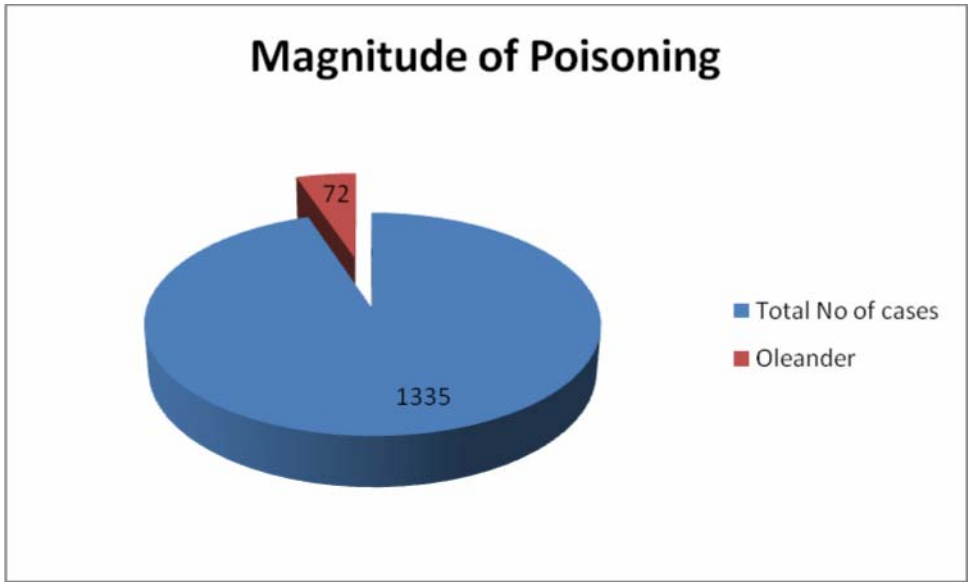



**Table: 7**

| SI NO | Cases | Frequency(n) |
|---|---|---|
| 1 | Total | 1335 |
| 2 | Oleander | 72 |

Oleander Seed poison cases accounted for 5% of the total Poison cases admitted.

**Fig: 8**

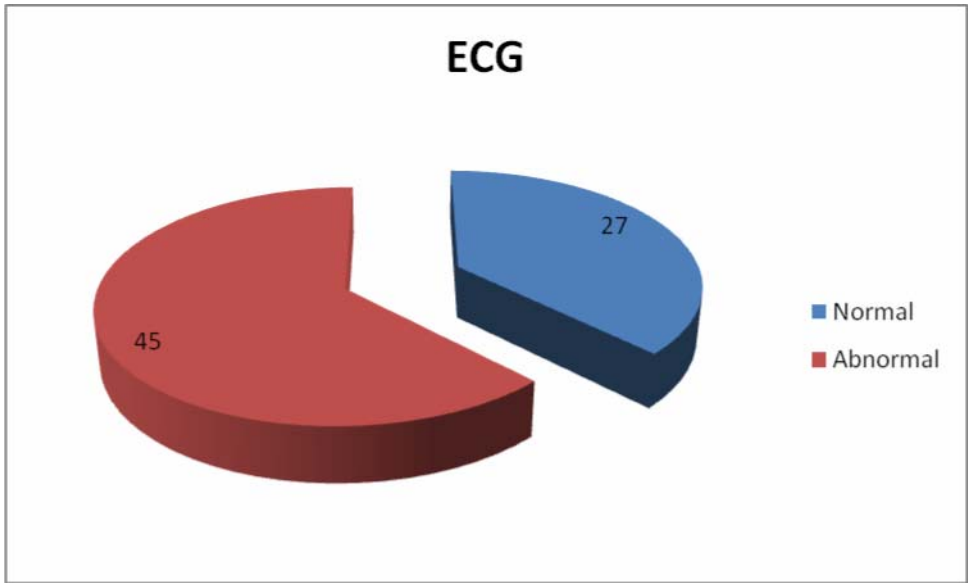
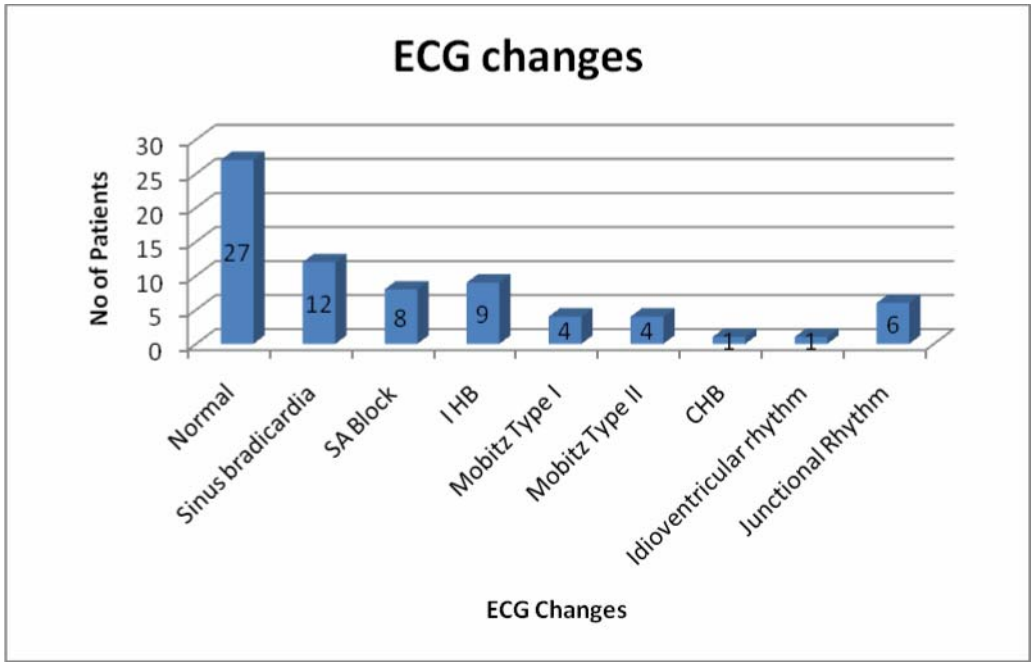



**Table: 8**

| SI NO | ECG | Frequency(n) | Percentage (%) |
|---|---|---|---|
| 1 | Normal | 27 | 37.5 |
| 2 | Abnormal | 45 | 62.5 |

More than 60% of the patients with Oleander poisoning had abnormal ECG's.

## Fig: 9




SA Block- Sino-Atrial Block, I HB- I Degree Heartblock,
CHB- Complete Heart Block.

## Table: 9

| SI No | ECG Changes | Frequency(n) | Percentage(%) |
|---|---|---|---|
| 1 | Normal | 27 | 37.5 |
| 2 | Sinus Bradicardia | 12 | 16.6 |
| 3 | Sino-Atrial Block | 8 | 11.11 |
| 4 | I Degree Hear Block | 9 | 12.5 |
| 5 | II Degree Heart Block<br>Mobitz Type I<br>Mobitz Type II | <br>4<br>4 | <br>5.5<br>5.5 |
| 6 | Complete heart block | 1 | 1.3 |
| 7 | Idioventricular Rhythm | 1 | 1.3 |
| 8 | Junctional Rhythm | 6 | 8.3 |

**Fig: 10**

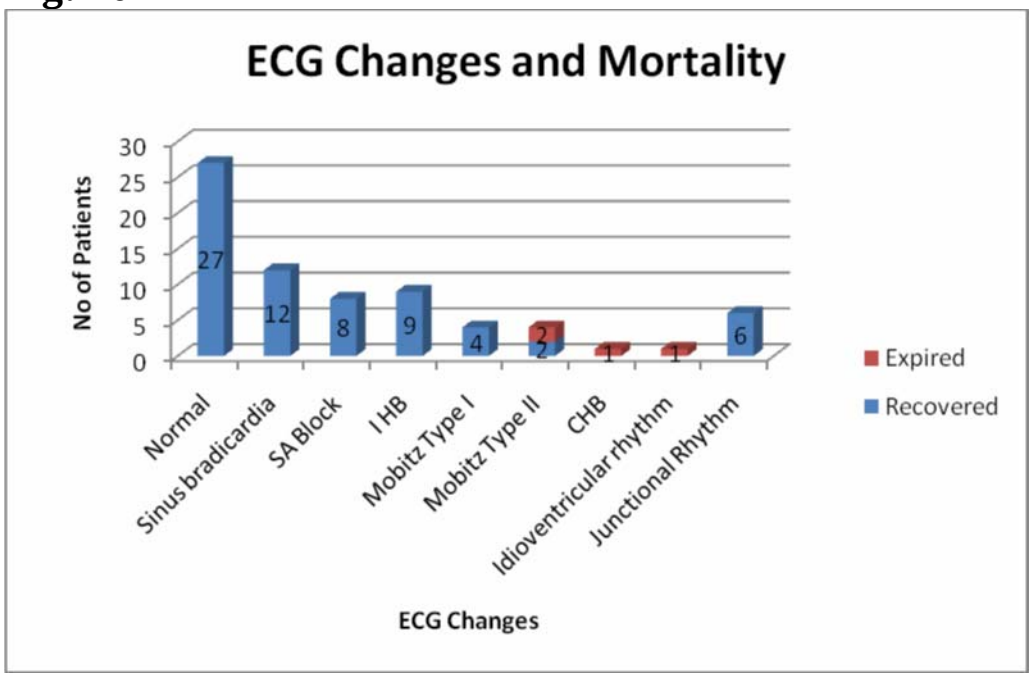



**Table: 10**

| SI No | ECG Changes | Frequency(n) | Death | Percentage(%) |
|---|---|---|---|---|
| 1 | Normal | 27 | 0 | 0 |
| 2 | Sinus Bradi | 12 | 0 | 0 |
| 3 | SinoAtrial Block | 8 | 0 | 0 |
| 4 | I HB | 9 | 0 | 0 |
| 5 | II Degree Heart Block<br>Mobitz Type I<br>Mobitz Type II | <br>4<br>4 | <br>0<br>2 | <br>0<br>2.7 |
| 6 | Complete heart Block | 1 | 1 | 1.38 |
| 7 | Idioventrricular Rhythm | 1 | 1 | 1.38 |
| 8 | Junctional Rhythm | 6 | 0 | 0 |

Among the four patients who died two of them had II Degree Heart Block (Mobitz type II), one had Idioventricular Rhythm and last one had complete heart block.

**Table 11: Bio Chemistry Values**

| SI.No | Values | Mean | S.D(+/-) |
|---|---|---|---|
| 1. | Blood Sugar mg/dl | 115.94 | 70.82 |
| 2. | Urea mg/dl | 24.97 | 9.15 |
| 3. | Creatinine | 0.85 | 6.18 |
| 4. | Sodium | 125.54 | 8.20 |

**Table 12: Liver Function Test**

| SI.No | Values | Mean | S.D(+/-) |
|---|---|---|---|
| 1. | Serum Bilirubin | 0.72 | 0.10 |
| 2. | AST | 24.24 | 14.32 |
| 3. | ALT | 22.4 | 10.0 |
| 4. | Total Protein g/dl | 6.5 | 0.50 |

**Fig : 11**

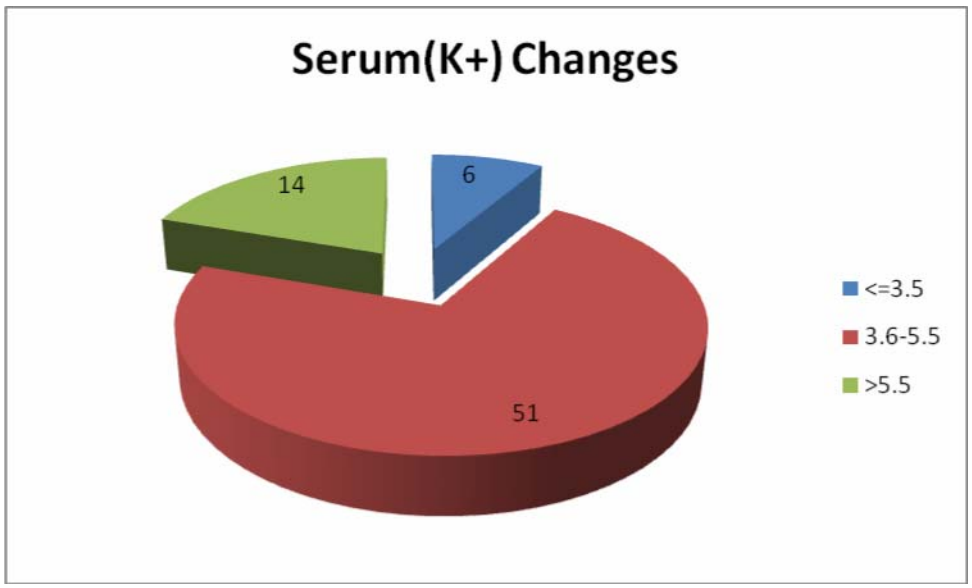



**Table: 13**

| SI NO | K+ Changes | Frequency(n) | Percentage(%) |
|---|---|---|---|
| 1 | <=3.5 | 6 | 8.4 |
| 2 | 3.6-5.5 | 51 | 71.8 |
| 3 | >5.5 | 14 | 19.7 |

Hyperkalemia presented in 19.44% of the patients with Oleander Seed Poisoning. 8.4% had Hypokalemia.

**Fig: 12**

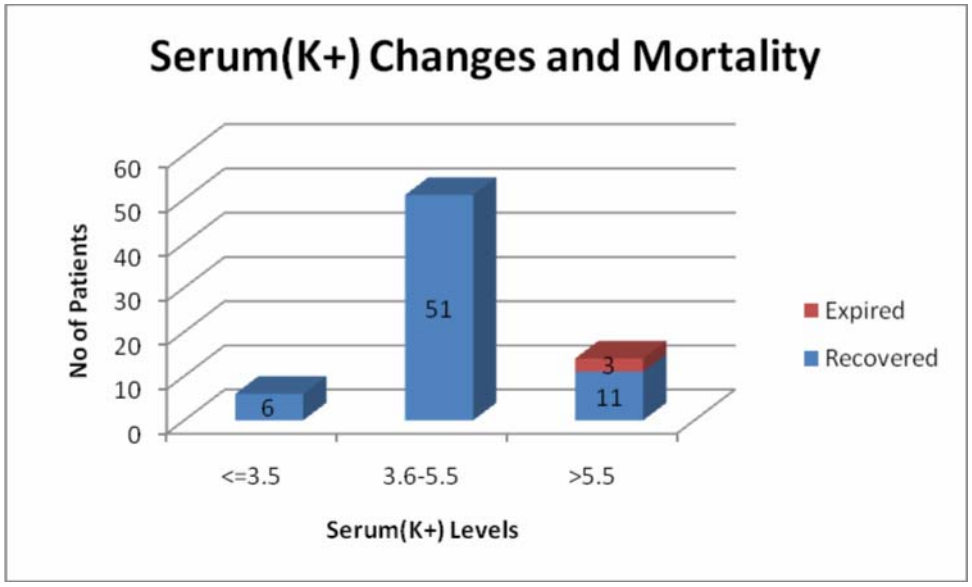



**Table: 14**

| SI NO | K+ Levels | Frequency(n) | Death | Percentage(%) |
|---|---|---|---|---|
| 1 | <=3.5 | 6 | 0 | 0 |
| 2 | 3.6-5.5 | 51 | 0 | 0 |
| 3 | >5.5 | 14 | 3 | 21.43 |

Because of early death (with in 35 min), serum electrolytes could not be measured, in one among the four patients who expired.

**Fig: 13**

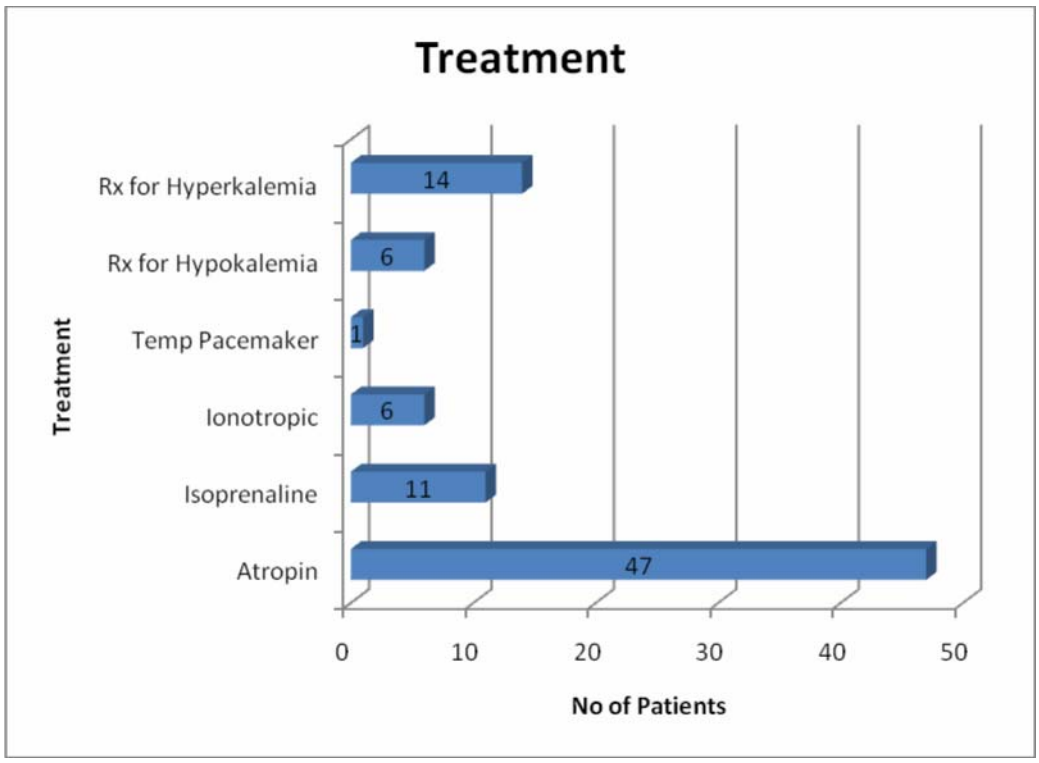

Treatment
Rx for Hyperkalemia
Rx for Hypokalemia
Temp Pacemaker
Ionotropic
Isoprenaline
Atropin
14
6
1
6
11
47
Treatment
0
10
20
30
40
50
No of Patients

**Fig: 14**

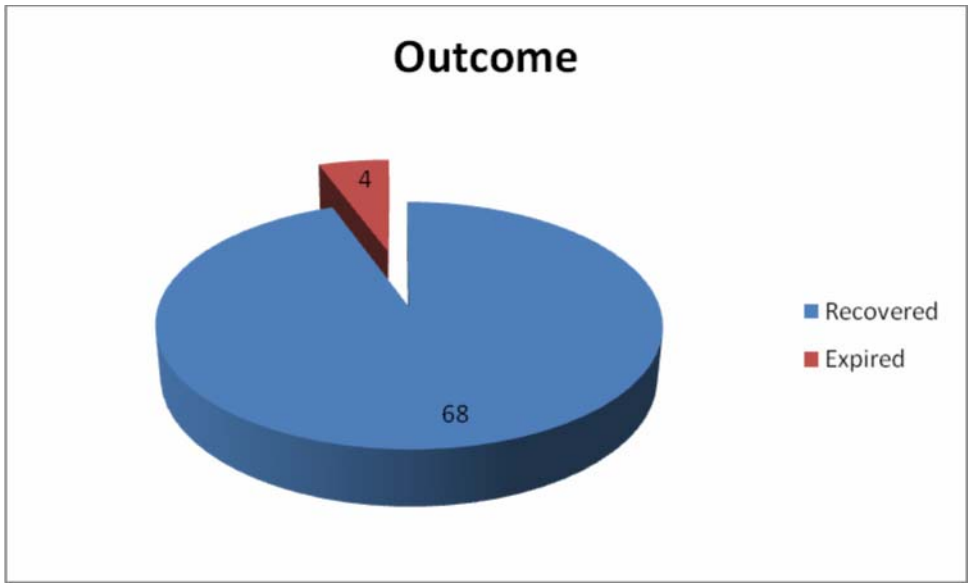



**Table : 15**

| SI NO | Outcome | Frequency(n) | Percentage(%) |
|---|---|---|---|
| 1 | Recovered | 68 | 94.4 |
| 2 | Expired | 4 | 5.6 |

Mortality rate among those who consumed Oleander seed as a poison was 5.6%.

**Tab 16: Analysis of Cases Expired**

| Case No. | Duration of Stay in Hrs | ECG Changes | Number of Seeds |
|---|---|---|---|
| 1 | 7 | Idioventricular Rhythm | 3 |
| 2 | 48 | Mobitz Type II | 4 |
| 3 | 4 | Mobitz Type II | 4 |
| 4 | 35 Min | Complete heart block | 10 |

## POSTMORTEM CHANGES

Postmortem was conducted to one patient it showed.

Section from heart shows normal cardiac muscle fibrosis with intervening interstium showing areas of hemorrhage. No evidence of coagulate necrosis is seen.

Section from aorta shows thickened media and adventia showing congested blood vessels.

Section from brain shows mild edema and congested blood vessels.

Section from liver shows sinusoidal congestion.

Section from kidney shows normal glomeruli with congestion of interstium; no significant tubular changes are seen.

## DISCUSSION

Comprehensive analysis of 72 cases of acute yellow oleander seed poisoning.

## EPIDEMIOLOGY OF ACUTE YELLOW OLEANDER SEED POISONING

### Age pattern

In this series of 72 cases, the number of the patients below 20 years of age were 19(26%), between 21-30 were 42(58%) and remaining were 11(16%). In Thanjavur by Bobby et al, number of patients below age group of 20 was 41.17% and between age group of 21-30 was 37.25% and remaining were 23%[38]. Eddleston et al of SRILANKA revealed that age range 12-77 years, median 21 years[25, 27, 29, 37]. The series reported in this study had the similar pattern of age group affection. The reason could be that this age group by all probability is vulnerable to various emotional conflicts that occur during demanding phase of life. This young age group affected by exposure forms the viable entity of any population both in terms of procurement and productivity. This case study and the case reports mentioned above throw light on the target age group for educative and preventive programs to reduce the incidence of oleander seed poisoning.

## SEX DISTRIBUTION

In poison center GGH Chennai females were exposed slightly more than male's population (51.4% versus 48.6%). In observation of 52 cases in Thanjavur by Bobby et al revealed that female (32), male (19) [38]. On the contrary in Srilanka by Eddleston et al in the observation of 1939 cases admitted males were 1021 (52.7%)[25,27,29,37]. This variation was due to handling of poison by the respective sex in their respective locality.

## MORTALITY AND AGE

Overall mortality in this study was 5 (5.5%). In age group of below 20 the mortality rate was 1(1.4%), between 20-30 was 2(2.7%) and 31-40 was 1(1.4%). In Thanjavur by Bobby et al in the observation of 51 cases the mortality rate was 1(1.96%) in the age group of 20-30[38]. In Srilanka by Eddleston et al in the observation of the 1939 patients (4.8%) were died. On contrary to our study, in Srilanka people over 64 years old were 13.8 (95%) times more likely to die than those less than of 25 years[25, 27, 29, 37]. In our study the number of patients in the age group of more than 40 years was five and mortality was nil.

## MORTALITY AND SEX

The percentage of mortality in males (8.5%) was higher compared to the females (2.71%). In Srilanka by Eddleston et al also similar findings were observed, the female case fatality was (3.9% vs. male 5.7%) [25,27,29,37].

## MAGNITUDE OF POISONING

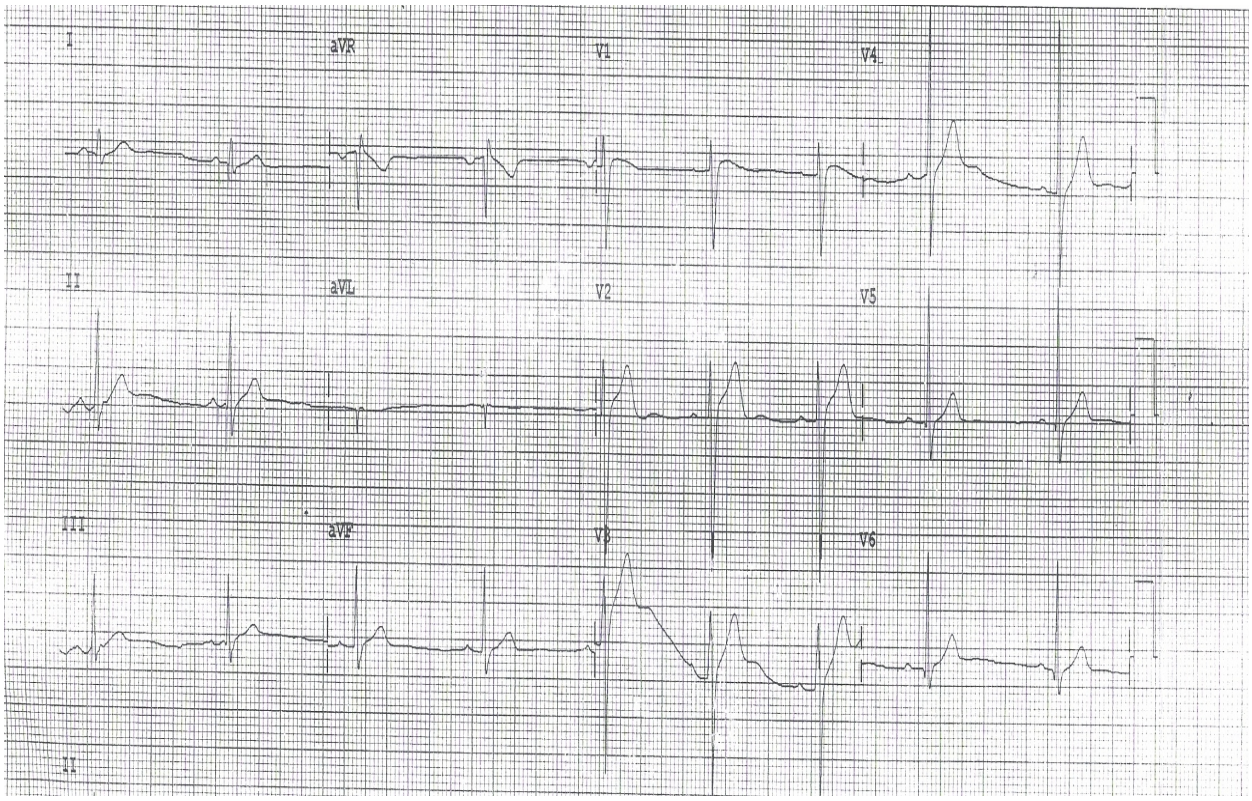
In our Toxicology, GGH in the period of six months the Magnitude of the Oleander seed poisoning admitted was 72(5%) from the total number of poison cases (1335) admitted. In these 72 cases 58(80.5%) cases were referred from near by GH and PHC's belonging to five districts (Chennai, Kancheepuram, Thiruvallur, vizhuppuram and Vellore).

## ECG ABNORMALITIES

Among the 72 patients, the significant ECG abnormalities were found in 45(62.5%), 37.5% had normal rate and rhythm. In these 16.6% had Sinus Bradycardia, 12.5% and 11% respectively had II Degree Heart Block & I. Among the 11% of the II Degree Heart Block 5.5% with Mobitz Type II Block. 1.3% had the complete Heart Block. 11.11% had SA block and 8.3% presented with Junctional rhythm. 1.3% had idioventricular rhythm and in our study,

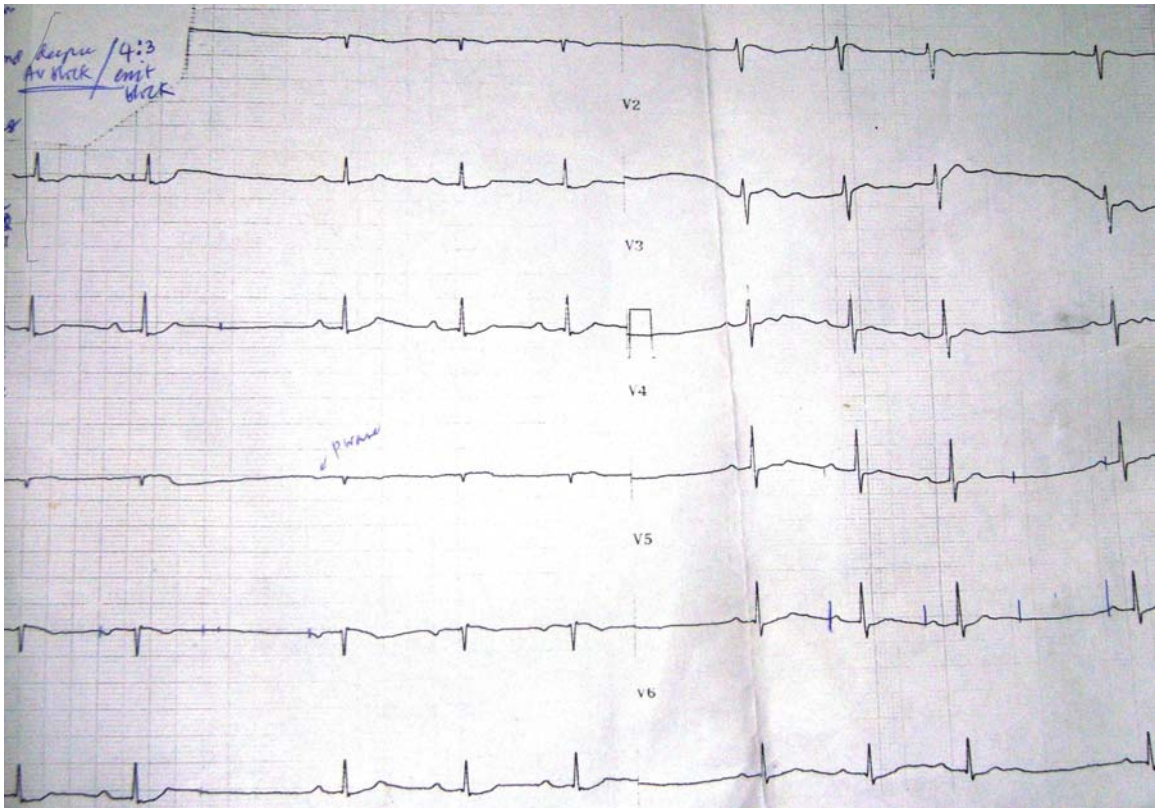
Ventricular ectopics and Bundle branch blocks were not found. In Srilanka by Eddleston et al was observed among 89 seriously ill patients, 53% had AV node conduction block, 62% had sinus node block; 30% had conduction block affecting both nodes, 1% had ventricular tachycardias and 8% had ventricular ectopics[25, 27, 29, 37]. In Thanjavur by Bobby et al, observed that 27 patients who had ECG abnormalities among the 51 patients, Sinus Bradycardia (59.25%), Sinus tachycardia(11.11%), T-wave inversion(7.4%), S-T depression(11.11%), SA Block(7.4%), I Degree Block(11.11%), II Degree Block(3.7%), and Ventricular Tachycardia(3.7%) [38].

**ECG 1**



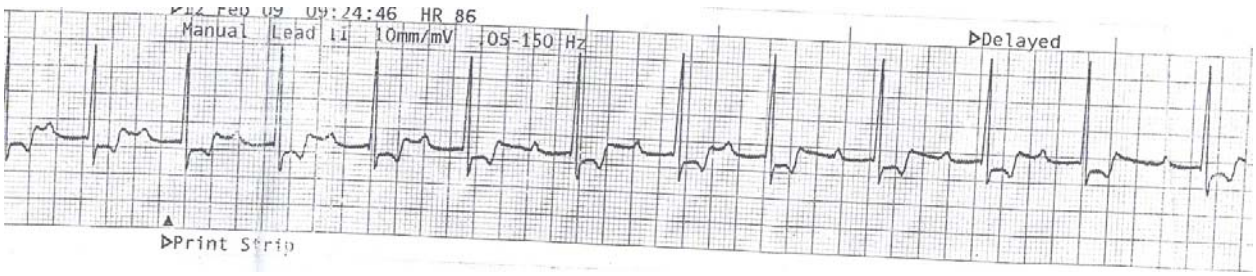

ECG 1:  shows Sinus Bradycardia (Heart Rate 50).

**ECG 2**



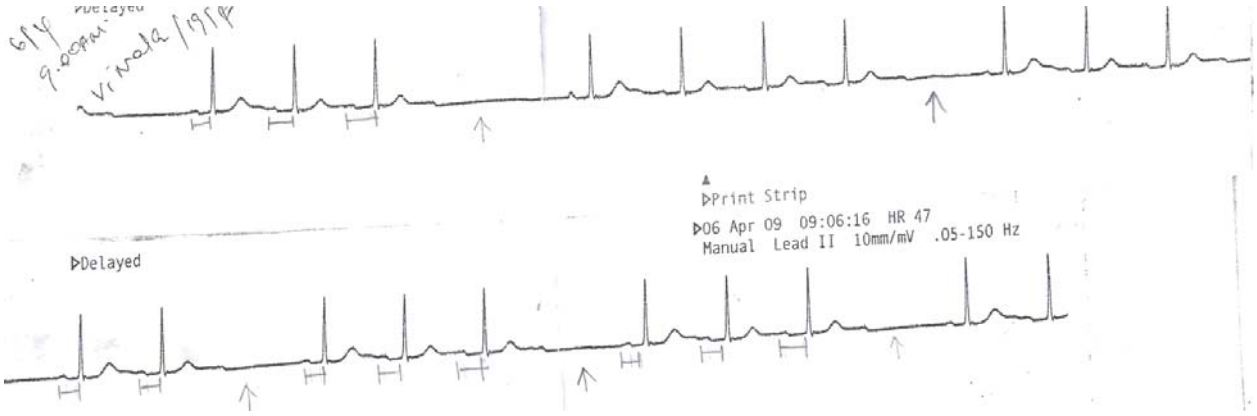

ECG 2 shows Sino-Atrial (SA) block. (No P wave or QRS complex is recorded drops out of complete cardiac cycle).

**ECG 3**



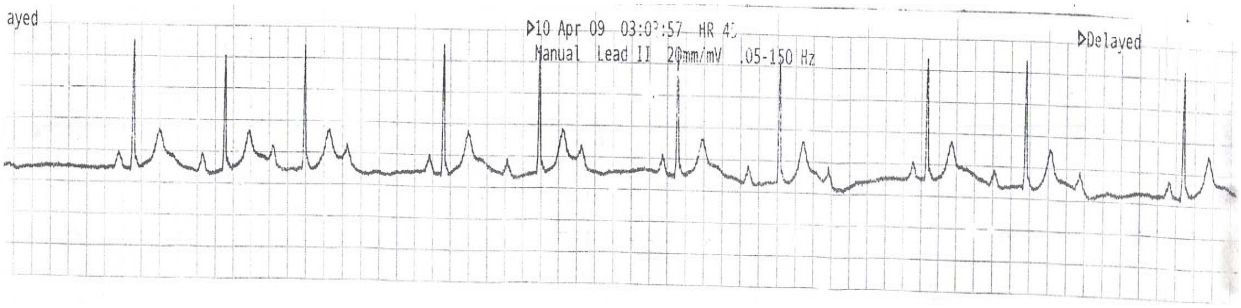

ECG 3: shows First Degree Heart Block (PR Interval more than 0.20s).

**ECG 4**



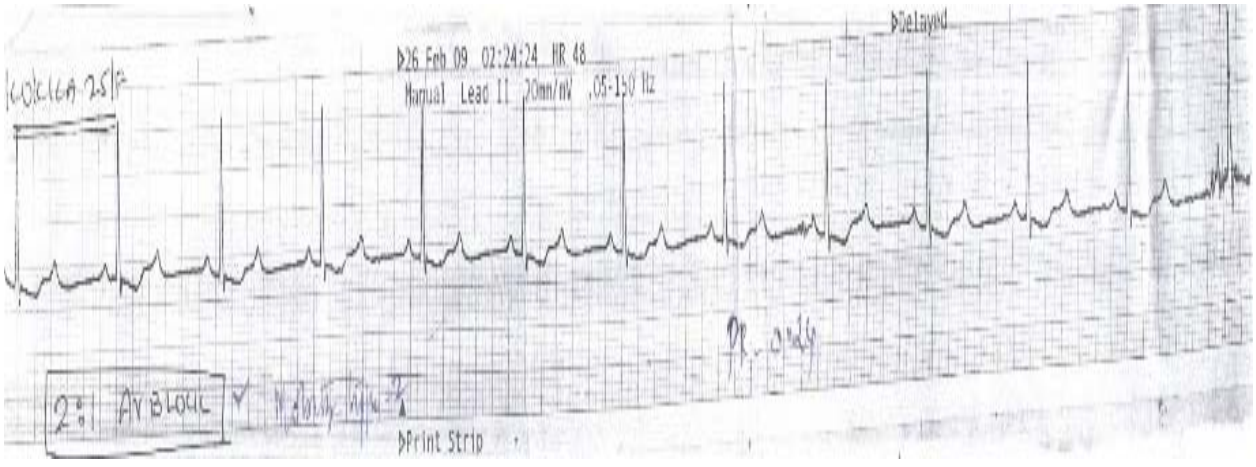

ECG 4: shows Mobitz Type I AV Block (Progressive lengthening of successive PR interval until P wave is not conducted) [36].

**ECG 5**



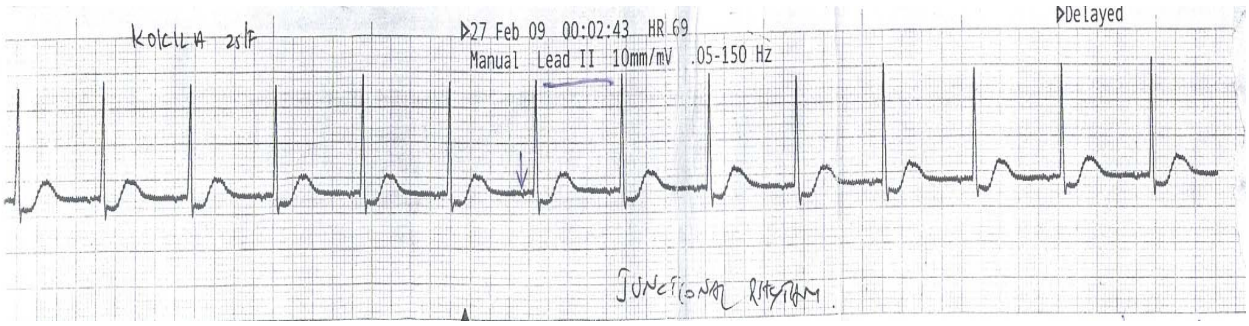

ECG 5: Shows Mobitz Type II 2:1 AV Block. In this P-R intervals of all conducted supra ventricular impulses are constant but alternate P wave is not conducted [35].

**ECG 6**



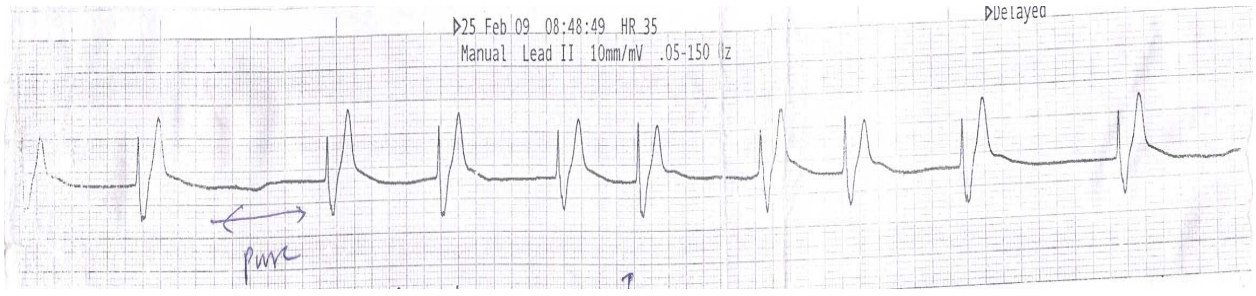

ECG 6: Shows similar finding as above ECG; Mobitz type II 2:1 AV Block.

**ECG 7**



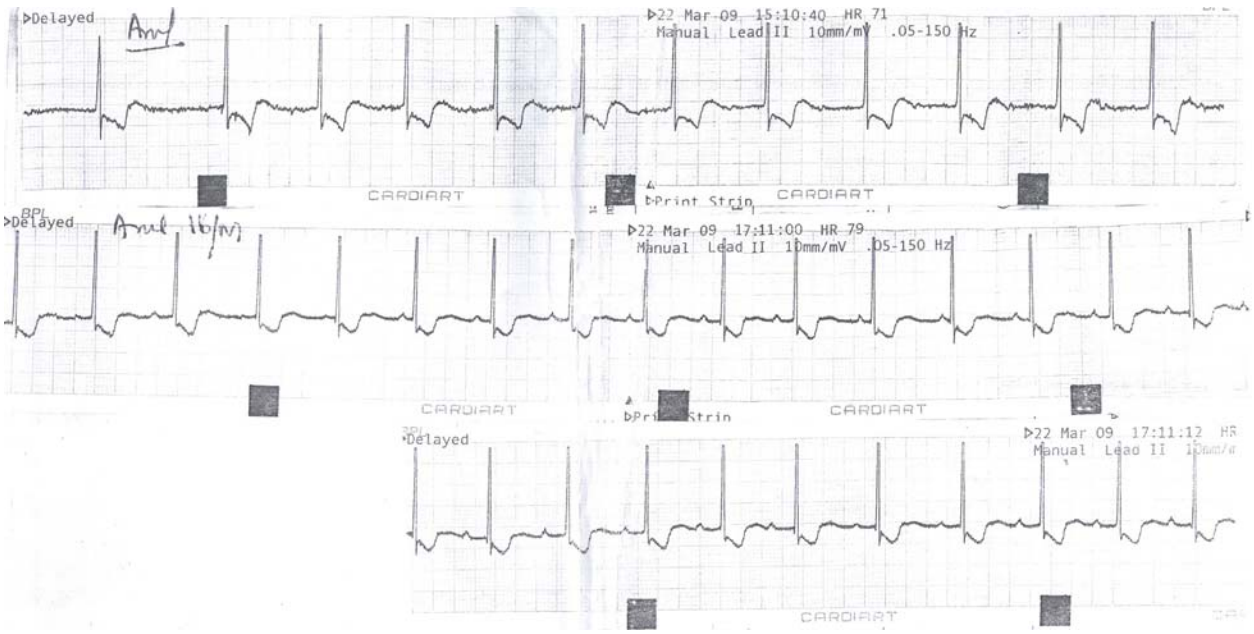

ECG 7: Taken from the same patient of ECG 5 shows Accelerated Junctional Rhythm (No proceding P waves, narrow QRS Complex, Rate 75).

**ECG 8**




ECG 8: Shows Accelerated Idio ventricular Rhythm (Frequent and wide QRS complexes not preceded by P waves [34].

**ECG 9**




ECG 9: shows evidence of Junctional Rhythm later reverted to Sinus Rhythm and shows ST-T changes similar as Digoxin effects ( Straight downward slope with terminal rise – mirror image of check mark ) [34,35].

## RENAL AND LIVER FUNCTION TEST

No significant abnormalities were found in Renal and Liver functions. Eddleston et al in Srilanka also noted similar observation[25, 27, 29, 37].

## ELECTROLYTES

No significant change in Serum Sodium level was noted. But significant change in the Serum Potassium levels was noted in 28.1% of patients. In this 19.7% had Hyper-Kalemia and 8.4% had Hypo-Kalemia. Among the 4 patients died in our series, 3 had Hyper-Kalemia. Remaining one patient went to cardiac arrest immediately after admission; So Laboratory investigation could not be done. Similarly studies conducted in Ananthapura in Srilanka by Eddleston et al revealed that the ECG changes and the mortality were higher in those who had Hyper-Kalemia. But exact incidence was not given.

## MANAGEMENT

In 72 patients, except one all of them underwent stomach wash and Activated Charcoal treatment. In this, 8 patients presented with normal rate and rhythm, doesn't need any active treatment. In the remaining 64 patients 7% of them required Ionotropic support, 15% required Isoprenaline support. Pacemaker support was given to 1 patient. 16% of the patients required management for Hyperkalemia, and 8% for Hypokalemia treatment.

**MORTALITY**

Overall mortality in our study was 4(5.5%). In the four patients who died in our study,

- One had Mobitz type II block consumed 4 seeds, shifted 14 hours after the consumption, and had a potassium level of 6 meq/l.
- One had Mobitz type II block and complex Arrhythmias, consumed 4 seeds, shifted 22 hours after the consumption, and had the potassium level of 7 meq/l.
- One had Junctional Rhythm with ST-T changes progressed to idioventricular rhythm with hypotension, consumed 3 seeds, and shifted after 24 hours of consumption, had the potassium level of 5.7 meq/l.
- One patient died because of Cardiac arrest with in 35 minutes of admission, shifted after 15 hrs of consumption, consumed 10 seeds. This patient had hypotension and complete heart block in ECG, which was taken in peripheral hospital prior to referral. Serum Electrolyte was not taken because of early death.

Mortality observed by Eddleston et al in Srilanka was 4.8%[25, 27, 29, 37]. In Thanjavur by Bobby et al observed that mortality was 2% due to ventricular

Tachycardia [38]. So deliberate self-poisoning is an important problem in the developing world, where the case fatality rate is far higher than in industrialized countries — 20% vs. <1% in the UK.[25]

One reason for this large difference is the lack of antidotes for many of the poisons mostly used in poor agricultural communities.

## CONCLUSION

1. Oleander seed is still used as a suicidal agent.
2. Oleander plant is easily available as an ornamental plant in urban, semi-urban and rural areas.
3. Oleander seed poison is most prevalent in the 14-30 age groups.
4. Incidence is almost equal among the genders.
5. Death of patients was independent of the number of seeds they consumed.
6. ECG abnormalities were found in majority of the individuals.
7. Electrolyte disturbances (Changes in serum K+ levels) were found in significant proportion of the patients.
8. Prognosis was poor among those who presenting with Hypo-tension, Electrolyte disturbances specially those with Hyperkalemia, and complex Arrhythmias.
9. The arrhythmias produced by this poisoning may range from Sinus bradycardia to complete heart block.

## STRENGTHS AND LIMITATIONS:

1. The limitations were single center study and children's were not included.

2. Serum cardiac Glycosides (Digoxin) levels were not tested for the patients.

3. Anti Digoxin-AB treatment was not given

## RECOMMENDATIONS

- Considering mortality and morbidity associated with Oleander poisoning, it may be recommended that the use of oleander as an ornamental plants be avoided.

- As there are no standard guidelines with reference to the indication for temporary pacemaker in the management of Oleander induced arrhythmias at present, uniform guidelines have to be formulated.

## BIBILIOGRAPHY

1. Pearn J. Oleander poisoning. In: Covacevich J, Davine P, Pearn J, eds. Toxic plants and animals: a guide for Australia, 2nd ed. Brisbane: William Brooks, 1989:37-50.

2. Langford SD, Boor PJ. Oleander toxicity: an examination of The human and animal toxic exposures. Toxicology 1996; 109:1-13.

3. Sreeharan N, Putharasingam S, Ranjadayalan K, et al. Yellow oleander poisoning—clinical manifestations and prognostic criteria. JaVna Med J 1985;20:100-1.

4. Saravanapavananthan N, Ganeshamoorthy J. Yellow Oleander poisoning—a study of 170 cases. Forensic Sci Int 1988; 36:247-50.

5. Fisch C, Surawicz B, eds. Digitalis. New York: Grune and Stratton, 1969.

6. Shaw D, Pearn J. Oleander poisoning. Med J Aust 1979; 2:267-9.

7. Brewster D. Herbal poisoning: a case report of a fatal yellow Oleander poisoning from the Solomon Islands. Ann Trop Pediatric 1986; 6:289-91.

8. Haynes BE, Bessen HA, Wightman WD. Oleander tea:

Herbal draught of death. Ann Emerg Med 1985; 14:350-3.

9. Eddleston M, Ariaratnam CA, Meyer PW, et al. Epidemic of Self-poisoning with seeds of the yellow oleander tree (Thevetia peruviana) in northern Sri Lanka. Trop Med Int Health 1999; 4:266-73.

10. Eddleston M, SheriV MHR, Hawton K. Deliberate Self-harm in Sri Lanka: an overlooked tragedy in the developing world. BMJ 1998; 317:133-5.

11. The Essentials of Forensic Medicine and Toxicology; Dr.K.S.Narayana Reddy, Twenty-fifth Edition 2006.

12. Nathanson JA. Caffeine and related methylxanthines: Possible naturally occurring pesticides. Science 1984; 226: 184-7.

13 Heywood VH, ed. Flowering plants of the world. Oxford: Oxford University Press, 1978.

14. API Textbook of Medicine, 8th Edition, Siddharth N.Shah, M.Paul Anand.

15. Ansford AJ, Morris H: Fatal oleander poisoning. Med J Aust 1981; 1:360-361

16. Osterloh J, Herold S, Pond S: Oleander interference in the digoxin radioimmunoassay in a fatal ingestion. JAMA 1982; 247:1596-1597

17. Shumaik GM, Wu AW, Ping AC: Oleander poisoning: Treatment with digoxin-specific Fab antibody fragments. Ann Emerg Med 1988; 17:732-735 .

18. Haynes BE, Bessen HA, Wightman WD: Oleander tea: Herbal draught of death. Ann Emerg Med 1985; 14:350-353

19. Shaw D, Pearn J: Oleander poisoning. Med J Aust 1979; 2:267-269

20. Mack RB: `To see a world in a grain of sand and a heaven in a wild flower'-Oleander poisoning. N C Med J 1984; 45:729-730

21. Kaojarern S, Sukhupunyarak S, Mokkhavesa C: Oleander Yee tho poisoning. J Med Assoc Thai 1986; 69:108-111

22. Mallick BK: Cardio toxicity in yellow oleander seed poisoning. I Indian Med Assoc 1984; 82:296-297

23. Cheung K, Hinds JA, Duffy P: Detection of poisoning by plant-origin cardiac glycoside with the Abbott TDX analyzer. Clin Chem 1989; 35:295-297.

24. Kelly RA, Smith TW. Recognition and management of digitalis toxicity. Am J Cardiol 1992; 69:108-19G.

25. Eddleston, C A Ariaratnam, K Rajakanthan, D Colbert, W P Meyer, G Warrell, Acute yellow oleander (Thevetia peruviana) poisoning: cardiac arrhythmias, electrolyte disturbances, and serum cardiac glycoside Concentrations on presentation to hospital, Heart 2000; 83:301-306.

26. Acute Oleander Poisoning A Suicide Attempt in a Geriatric Patient David A. Driggers, MD Ronald Solbrig, MD Joseph F steiner, PharmD, Jay Swedberg, MD Gary S. Jewell, MD Casper, Wyoming.

27. Eddleston M, Sheriff MHR, Hawton K. Deliberate self-harm in Sri Lanka: an overlooked tragedy in the developing world. Br Med J 1998; 317:133–5

28. Sreeharan et al. 1985; Saravanapavananthan & Ganeshamoorthy 1988).

29. Epidemic of self-poisoning with seeds of the yellow oleander tree (Thevetia peruviana) in northern Sri Lanka, M. Eddleston1, C.A. Ariaratnam2, W. P.Meyer3,G. Perera1, A.M.Kularatne4, S. Attapattu4,M.H. R. Sheriff2 and D.A.Warrell1.

30. Qualitative Identification of some toxic plants constituent. Prepared By: Mosa Qasheesh.

31. Source: Modified from Gregoratos et al, 1997, and Gregoratos et al, 2002.

32. Harrision, Principle of Internal Medicine, Edition 17.

33. Lippincott's Illustrated Reviews: Pharmacology, 4th Edition: Finkel, Richard; Clark, Michelle A.; Cubeddu, Luigi X.

34. Leoschamroth, An introduction to electrocardiography 7 th edition: revised by colinschamroth.

35. Marriotts Practical Electrocardiography, 11th edition, Galen S.Wagner.

36. Davidsons Principles and Practice of medicine, 20th edition, by Christopher Haslett.

37. Q J Oxford journal of Med 1999; 92: 483-485 © 1999, Management of acute yellow oleander poisoning, M. Eddleston and D.A. Warrell. Centre for Tropical Medicine, Nuffield Department of Clinical Medicine, University of Oxford.

38. Epidemiological profile of Oleander seed poisoning in Thanjavur by Bobby et all.

39. Donald B.Kennel and David G.Apreke 1984 – Emergency Medicine clinics of North America- Vol 2, No.1 Feb 1984.

40. Gold frank's Toxicological Emergencies – 8th Edition - 2006.

# INSTITUTIONAL ETHICAL COMMITTEE
## MADRAS MEDICAL COLLEGE,CHENNAI-600 003.

Telephone : 25363970

Fax :044 – 253-5115
:044 25363970

L.Dis.No. 14597/ME5/EthicsDean/MMC/2009 Dated .09.2009

Title of the work : "To analyse cardiac arrythmias and electro
Principal Investigator : disturbances in patients with acute yello
oleander poisoning.

Department : Dr. M. Sudumathi P.G - M.D (Internal medici
madras medical college - ch - 3

The request for an approval from the Institutional Ethical Committee(IEC) was considered on the IEC meeting held on 23rd September 2009 at 2.00P.M. in Madras Medical College, Deans, Chamber, Chennai-3./ Pharmacology seminar hall - madras medical college ch

The members of the Committee, the Secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The principal investigator and their term are directed to adhere the guidelines given below:

1. You should get detailed informed consent from the patients/participants and maintain confidentiality.
2. You should carry out the work without detrimental to regular activities as well as without extra expenditure to the Institution or Government.
3. You should inform the IEC in case of any change of study procedure, site and investigation or guide.
4. You should not deviate form the area of the work for which I applied for ethical clearnance.
5. You should inform the IEC immediately, in case of any adverse events or serious adverse reactions.
6. You should abide to the rules and regulations of the institution(s).
7. You should complete the work within the specific period and if any extension of time is required, you should apply for permission again and do the work.
8. You should submit the summary of the work to the ethical committee on completion of the work.
9. You should not claim funds from the Institution while doing the work or on completion.
10. You should understand that the members of IEC have the right to monitor the work with prior intimation.

SECRETARY
IEC, MMC,CHENNAI

CHAIRMAN
IEC MMC CHENNAI

DEAN
MADRAS MEDICAL COLLEGE
CHENNAI

**PROFORMA**

NAME

AGE

SEX

OCCUPATION

LOCATION

DATE OF ADMISSION

DATE OF SAMPLING

ADMISSION DIAGNOSIS

BRIEF HISTORY

NO OF SEEDS AND LEAVES CONSUMED

Date of ingestion

Time of ingestion

Time since consumption

Symptoms on presentation in detail:

Prior treatment:

Provider:

Supportive:

Antidote:

Time taken to reach first treatment centre:

Personal history :

Previous history of poisoning [yes / no] if yes _details:

History of alcohol and drug abuse:if yes details:

High risk behavior:

Family history of poisoning [yes/no] if yes details:

PHYSICAL EXAMINATION

GENERAL EXAMINATION

VITAL SIGNS

PULSE DAY 1 DAY 2 DAY3 DAY 5

Rate

Rhythm

BP

RESPIRATORY RATE

RECTAL TEMPERATURE

CARDIOVASCULAR SYSTEM

RESPIRATORY SYSTEM

GASTROINTESTINAL SYSTEM

CENTRAL NERVOUS SYSTEM

GCS

INVESTIGATIONS

CBC

TC

DC

PCV

ESR

PLATELETS

RFT

| | DAY 1 | DAY 2 | DAY 3 |
|---|---|---|---|
| DAY 5 | | | |
| BLOOD SUGAR | | | |
| UREA | | | |
| SERUM CREATININE | | | |
| Na+ | | | |
| K+ | | | |

LFT

TOTAL BILIRUBIN

AST

ALT

SAP

TOTAL PROTEIN

ABG

PH

PO2

PCO2

SAO2

HCO3

Pao2/ Fio2 ratio

| ECG | DAY 1 | DAY2 | DAY3 | DAY5 |
|---|---|---|---|---|
| RATE | | | | |
| RHYTHM | | | | |
| P wave | | | | |
| P-R interval | | | | |
| QRS complex | | | | |
| Width | | | | |
| Axis | | | | |
| Configuration | | | | |
| S-T segment | | | | |
| T wave | | | | |
| U wave | | | | |
| Comments | | | | |

TREATMENT

FINAL OUTCOME

IF DEATH- POST MORTEM REPORT:

IMPRESSION:

# LIST OF TABLES

# LIST OF FIGURES

| NAME | SEX | AGE | LOCATION | DOA | T O A | NO OF SEEDS | D O I | T O I | T S C | R OR NOR |
|---|---|---|---|---|---|---|---|---|---|---|
| Alamelu | F | 22 | Kancheepuram | 7/3/2009 | 01.30 PM | 3 Seeds | 06/03/09 | 02.00 PM | 24 Hrs | R |
| Anjali | F | 20 | Chengalpat | 19/01/2009 | 11.00 PM | 4 Seeds | 19/01/2009 | 10.00 AM | 12 Hrs | R |
| kalaiselvi | F | 18 | kancheepuram | 3/2/2009 | 10.30 PM | 4 Seeds | 02/02/09 | 04.00 PM | 36 Hrs | R |
| kamakshi | F, P | 22 | Kaladipet | 9/2/2009 | 07.15 PM | 1 Seed | 09/02/09 | 02.00 PM | 5 Hrs | NR |
| Kamakshi 1 | F | 18 | Thiruvallur Dt | 15/01/2009 | 04.00 PM | 4 Seeds | 15/01/2009 | 9.00 AM | 7 Hrs | R |
| Loganayagi | F | 29 | Thiruvallur Dt | 15/02/2009 | 11.30 PM | 1 Seed | 15/02/2009 | 7.00 PM | 4 Hrs | R |
| Sarawathi | F | 30 | Chindadripet | 16/01/2009 | 02.25 AM | 1 Seed | 16/01/2009 | 10.00 PM | 4 Hrs | NR |
| Sathish | M | 19 | Thiruvallur Dt | 22/01/2009 | 01.24 PM | 10 Seeds | 22/01/2009 | 08.00 AM | 6 Hrs | R |
| Sekar | M | 35 | Thiruvallur Dt | 11/2/09 | 2.15 PM | 4 Seeds | 10/02/09 | 03.00 AM | 12 Hrs | R |
| Velankanni | F | 30 | Egmore | 9/1/2009 | 10.15 AM | 1 Seed | 09/01/09 | 9.00 AM | 1 Hrs | NR |
| Ammu Rajalakshmi | F | 14 | Pallavaram | 11/03/09 | 05.00 PM | 6 Seeds | 11/03/09 | 09.30 Am | 7 Hrs | R |
| Anjali | F | 18 | kancheepuram Dt | 4/4/2009 | 04.00 PM | 4 seeds | 03/04/09 | 3.00 PM | 23 Hrs | R |
| Desinga Raja | M | 22 | Thiruvallur Dt | 27/02/2009 | 03.10 PM | 1 Seed | 26/02/2009 | 01.00 PM | 36 Hrs | R |
| Gomathi | F,P | 21 | Kancheepuram Dt | 7/3/2009 | 08.30 PM | 1 Seed | 07/03/09 | 09.00 AM | 12 Hrs | R |
| Lakshmi | F | 22 | Thiruvallur Dt | 5/4/2009 | 04.20 PM | 2 Seeds | 05/04/09 | 01.00 PM | 3 Hrs | R |
| Purusothaman | M | 19 | Kancheepuram Dt | 19/03/2009 | 08.30 AM | 8 Seeds | 18/03/2009 | 02.00 AM | 28 Hrs | R |
| RoseMary | F | 23 | KK Nagar | 10/3/09 | 09.00 PM | 1 Seed | 10/03/09 | 06.30 PM | 2.30 Hrs | R |
| Samundi | M | 43 | Thiruvallur Dt | 4/4/2009 | 01.40 PM | 3 Seeds | 04/04/09 | 06.00 AM | 7 Hrs | R |
| Seetha | F | 18 | Thambaram | 18/02/2009 | 01.25 PM | 2 Seeds | 17/02/2009 | 07.00 AM | 32 Hrs | R |
| Vimala | F | 19 | Villupuram | 4/4/2009 | 02.42 PM | 4 Seeds | 03/04/09 | 1.00 PM | 25 Hrs | R |
| Anandhan | M | 26 | Kancheepuram | 25/02/2009 | 06.42 AM | 10 Seeds | 24/02/2009 | 4.00 PM | 14 Hrs | R |
| Arul | M | 16 | Kancheepuram Dt | 22/03/2009 | 01.10 PM | 4 Seeds | 21/03/2009 | 03.00 PM | 22 Hrs | R |
| Babu | M | 22 | Chindadripet | 5/3/2009 | 09.22 PM | 4 Seeds | 05/03/09 | 2.00 PM | 7 Hrs | NR |
| Nirmala | F | 21 | MGR Nagar | 13/04/2009 | 02.20 PM | 1 Seed | 13/04/2009 | 08.00 AM | 6 Hrs | R |
| Raja | M | 27 | Kolathur | 13/05/2009 | 02.30 PM | 3 Seeds | 13/05/2009 | 10.00 AM | 4 Hrs | NR |
| Rajendran | M | 52 | Villivakkam | 13/04/2009 | 10.30 AM | 4 Seeds | 12/04/09 | 07.00 PM | 15 Hrs | R |
| Rajesh | M | 23 | kancheepuram Dt | 27/02/2009 | 06.29 PM | 8 Seeds | 25/02/2009 | 03.14 PM | 48 Hrs | R |
| Tamilselvi | F | 29 | Chrompet | 6/3/2009 | 09.15 PM | 5 Seeds | 06/03/09 | 10.00 AM | 11 Hrs | R |
| Usha | F | 21 | Ambattur Estate | 10/4/09 | 01.50 AM | 2 Seeds | 09/04/09 | 03.00 PM | 13 Hrs | NR |
| Vatchala | F | 45 | kancheepuram Dt | 25/03/2009 | 07.45 PM | 2 Seeds | 25/03/2009 | 11.30 AM | 8 Hrs | R |
| Gajendran | M | 43 | kancheepuram Dt | 14/03/2009 | 09.05 AM | 3 Seeds | 14/03/2009 | 04.30 AM | 5 Hrs | R |
| George | M | 33 | Madhuranthagam | 21/02/2009 | 11.10 PM | 10 Seeds | 21/02/2009 | 07.00 AM | 16 Hrs | R |
| Kamatchi 2 | F | 31 | Poonamalle | 21/04/2009 | 01.50 AM | 3 Seeds | 20/04/2009 | 08.00 PM | 14 Hrs | NR |
| Gokila | F | 20 | Moongileri | 26/02/2009 | 12.30 AM | 10 Seeds | 25/02/2009 | 10.00 AM | 26 Hrs | R |
| Mohan | M | 22 | Kancheepuram | 15/04/2009 | 12.50 AM | 6 Seeds | 14/04/2009 | 09.00 PM | 3 Hrs | R |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Bhuvaneshwari | F | 25 | Thiruvallur Dt | 10/05/09 | 4.40 PM | 3 Seeds | 10/05/09 | 10.00 AM | 6 Hrs | R |
| Bhuvaneshwari | F | 16 | Arakkonam | 3/1/2009 | 11.40 PM | 4 Seeds | 03/01/09 | 09.00 AM | 14 Hrs | R |
| Dayalan | M | 30 | Velachery | 11/05/09 | 04.45 AM | 3 Seeds | 10/05/09 | 06.30 PM | 11 Hrs | NR |
| Gunasekar | M | 25 | Kancheepuram | 14/01/2009 | 01.00 AM | 6 Seeds | 13/01/2009 | 08.00 PM | 5 Hrs | R |
| Kavitha | F | 17 | Pallikaranai | 27/04/2009 | 11.25 PM | 1 Seed | 27/04/2009 | 12.00 PM | 11 Hrs | NR |
| Kosalai | F | 17 | Kancheepuram | 6/1/2009 | 03.00 PM | 1 Seed | 29/04/09. | 1.00pm | 6hrs | R |
| Malar | F | 25 | Kancheepuram Dt | 10/02/09 | 03.40 AM | 4 Seeds | 09/02/09 | 09.00 AM | 18 Hrs | R |
| Mari | M | 27 | Kancheepuram | 12/02/09 | 01.20 PM | 4 Seeds | 11/02/09 | 07.30 PM | 17 Hrs | R |
| Manivannan | M | 20 | Villupuram | 25/01/2009 | 06.25 PM | 4 Seeds | 25/01/2009 | 07.30 AM | 11 Hrs | R |
| Raja | M | 23 | Mangadu | 3/5/2009 | 03.10 AM | 4 Seeds | 02/05/09 | 05.30 PM | 9 Hrs | R |
| Ramesh | M | 22 | Thirunindravoor | 18/02/2009 | 11.24 PM | 1 Pulp of Seed | 18/02/2009 | 8.00 PM | 3 Hrs | NR |
| Sakthivel | M | 20 | mount | 10/06/09 | 12.24 PM | 3 Seeds | 10/06/09 | 09.00 AM | 3 Hrs | NR |
| Sarawathi | F | 30 | Thiruvallur Dt | 16/01/2009 | 02.10 AM | 1 Seed | 16/01/2009 | 08.00 PM | 6 Hrs | R |
| Srinivasan | M | 25 | Narthanarthapuram | 7/2/2009 | 02.20 PM | 4 Seeds | 07/02/09 | 09.00 AM | 5 Hrs | R |
| Subhalakshmi | F | 17 | Thiruvallore | 14/05/2009 | 12.15 PM | 5 Seeds | 13/05/2009 | 06.00 PM | 18 Hrs | R |
| Suresh | M | 26 | Thiruvallore | 25/05/2009 | 08.20 PM | Handfull of Seeds | 24/05/2009 | 08.20 PM | 24 Hrs | R |
| Thirupathi | M | 27 | Nanganallur | 19/02/2009 | 12.46 AM | 3 Pulp | 18/02/2009 | 7.00 PM | 5 Hrs | R |
| Udhayakumar | M | 20 | St.Thomas Mount | 2/5/2009 | 02.18 AM | 3 Seeds | 01/05/09 | 11.00 PM | 3 Hrs | NR |
| Vatchala | F | 18 | Villupuram | 8/3/2009 | 10.25 PM | 5 Seeds | 07/03/09 | 11.00 AM | 35 Hrs | R |
| Velankanni | F | 27 | Neelankarai | 15/02/2009 | 10.30 PM | 7 Seeds | 15/02/2009 | 07.00 PM | 3.30 Hrs | R |
| Velmurugan | M | 22 | Kancheepuram | 7/5/2009 | 05.21 PM | 10 Seeds | 05/05/09 | 10.30 AM | 48 Hrs | R |
| Viji | F | 17 | Kotturpuram | 12/06/09 | 05.11 PM | 4 Seeds | 08/06/09 | 06.30 PM | 4 Days | R |
| Senthil | M | 20 | Villupuram | 5/4/2009 | 04.20 AM | 4 Seeds | 04/04/09 | 08.00 AM | 20 Hrs | R |
| Gopi | M | 32 | Anagapalli, Chennai | 21/05/2009 | 03.47 AM | 7 Seeds | 20/05/2009 | 07.00 PM | 8 Hrs | NR |
| PonMeenakshi | F | 24 | thiruvallore | 16/01/09 | 6.00 pm | 5 Seeds | 20/01/2009 | 3.00 pm | 6hrs | R |
| Sathya | F | 18 | kancheeuram | 29/05/2009 | | 3 Seeds | 04/05/09 | 4.00pm | 7 hrs | R |
| Senthil | M | 30 | arakkonam | 29/06/2009 | | 4 Seeds | 06/05/09 | 8.00am | 2hrs | R |

| PR | BP | RBS | UREA | S.CR | NA+ | K+ | TB | AST | ALT | TP | Stay | Outcome | Atropine | Isoprenline |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 62 | 90/70 | 99 | 25 | 1 | 125 | 5.9 | 1 | 36 | 40 | 6 | 7 Hrs | D | Y | Yes |
| 92 | 90/60 | 105 | 37 | 1.1 | 135 | 4.9 | 1 | 53 | 27 | 6 | 4 Days | | Y | |
| 52 | 100/70 | 77 | 37 | 1 | 138 | 4.1 | 0.9 | 98 | 25 | 7 | 2 Days | | Y | |
| ## | 110/70 | 70 | 19 | 0.8 | 112 | 3.4 | 0.9 | 42 | 32 | 6 | 2 Days | | N | |
| 90 | 110/60 | 94 | 16 | 0.7 | 128 | 4.1 | 0.8 | 12 | 34 | 6 | 3 Days | | N | |
| 80 | 100/70 | 114 | 26 | 0.6 | 125 | 2.7 | 0.9 | 14 | 14 | 6 | 2 days | | Y | |
| 80 | 110/80 | 125 | 28 | 0.8 | 131 | 4.2 | 0.7 | 24 | 24 | 6 | 2 Days | | N | |
| 60 | 100/60 | 72 | 18 | 0.7 | 123 | 3.6 | 0.7 | 34 | 18 | 6 | 2 Days | | N | |
| 62 | 90/60 | 70 | 26 | 0.8 | 126 | 3.9 | 1.2 | 46 | 28 | 6 | 2 Days | | N | |
| 78 | 140/80 | Low | 19 | 0.6 | 143 | 4 | 0.7 | 18 | 14 | # | 2 Days | | N | |
| 90 | 110/70 | 132 | 21 | 0.9 | 129 | 3.8 | 0.8 | 27 | 32 | 7 | 4 Days | | Y | |
| 75 | 120/80 | 175 | 30 | 0.9 | 122 | 3.9 | 0.6 | 21 | 17 | 7 | 3 Days | | Y | |
| 64 | 110/80 | 88 | 20 | 0.8 | 140 | 4.5 | 1 | 31 | 27 | 7 | 2 Days | | N | |
| 65 | 120/80 | 156 | 26 | 0.9 | 128 | 4.3 | 0.8 | 24 | 27 | 7 | 3 Days | | N | |
| 60 | 100/60 | 70 | 20 | 0.7 | 128 | 3.9 | 0.7 | 18 | 15 | 6 | 2 Days | | N | |
| 88 | 120/80 | 68 | 23 | 0.8 | 127 | 5 | 0.9 | 20 | 15 | 6 | 4 Days | | Y | Yes |
| 78 | 110/70 | 119 | 16 | 0.7 | 123 | 3.8 | 0.7 | 30 | 19 | 4 | 3 Days | | N | |
| 78 | 110/90 | 87 | 3.1 | 0.9 | 140 | 4.3 | 0.8 | 28 | 26 | 7 | 3 Days | | N | |
| ## | 140/90 | 60 | 25 | 0.9 | 120 | 3.4 | 1 | 36 | 28 | 6 | 3 Days | | N | |
| 90 | 90/70 | 124 | 37 | 1 | 138 | 4.2 | 0.8 | 14 | 15 | 7 | 4 Days | | Y | Yes |
| 74 | 150/90 | 294 | 21 | 0.7 | 125 | 6 | 0.9 | 15 | 52 | 7 | 4 Hrs | D | Y | Yes |
| 70 | 90/70 | 91 | 39 | 1 | 146 | 5.9 | 0.8 | 25 | 30 | 6 | 2 Days | D | Y | Yes |
| 94 | 160/120 | 89 | 27 | 1 | 145 | 6 | 0.9 | 27 | 19 | 7 | 6 Days | | Y | |
| 80 | 110/70 | 77 | 21 | 0.9 | 125 | 2.3 | 0.9 | 19 | 27 | 6 | 4 Days | | N | Yes |
| 80 | 110/70 | 67 | 25 | 0.9 | 131 | 3.3 | 1 | 31 | 42 | 7 | 2 Days | | Y | |
| 80 | 100/70 | 186 | 24 | 0.9 | 132 | High | 0.8 | 16 | 23 | 6 | 5 Days | | Y | Yes |
| 44 | 120/80 | 141 | 25 | 1.1 | 124 | 3.2 | 0.9 | 23 | 29 | 6 | 5 Days | | Y | |
| 86 | 130/80 | 117 | 21 | 0.9 | 127 | 3.5 | 0.9 | 30 | 34 | 6 | 2 Days | | Y | |
| 82 | 100/60 | 335 | 74 | 1 | 132 | 5.7 | 0.8 | 23 | 21 | 6 | 5 Days | | Y | Yes |
| 80 | 110/80 | 67 | 22 | 0.9 | 147 | 3.4 | 0.8 | 22 | 30 | 6 | 2 Days | | N | |
| 88 | 110/80 | 116 | 17 | 0.6 | 146 | 4.4 | 0.9 | 37 | 50 | 4 | 2 Days | | N | |
| | | | | | | | | | | | 35 Mins | D | Y | |
| 90 | 120/70 | 79 | 26 | 0.6 | 149 | 3 | 1 | 18 | 14 | 7 | 2 Days | | Y | |
| 56 | 100/60 | 170 | 29 | 1 | 138 | 6 | 0.8 | 34 | 29 | 7 | 4 Days | | Y | Yes |
| 56 | 110/70 | 87 | 25 | 0.9 | 119 | 4.1 | 0.8 | 40 | 22 | 6 | 5 Days | | N | Yes |

| | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 42 | 13O/90 | 104 | 18 | 0.7 | 140 | 4.4 | TB | 50 | 32 | 6 | 4 | Days | Y | Yes |
| ## | 150/80 | 92 | 21 | 0.9 | 129 | 5.6 | 1 | 32 | 21 | 6 | 3 | Days | Y | |
| 70 | 120/80 | 103 | 20 | 0.9 | 128 | 5.4 | 1 | 18 | 20 | 6 | 3 | Days | N | |
| 88 | 140/90 | 87 | 28 | 0.8 | 127 | 3.6 | 0.9 | 45 | 33 | 6 | 4 | Days | Y | |
| 46 | 100/70 | 132 | 28 | 0.9 | 146 | 3.7 | 0.9 | 62 | 19 | 7 | 4 | Days | Y | Yes |
| 64 | 100/80 | 95 | 23 | 0.9 | 123 | 4 | 0.8 | 30 | 27 | 7 | 2 | Days | N | |
| 84 | 110/70 | 68 | 23 | 0.8 | 134 | 3.8 | 0.9 | 29 | 83 | 7 | 2 | Days | Y | |
| ## | 110/70 | 129 | 29 | 1 | 137 | 5 | 0.7 | 36 | 56 | 8 | 2 | Days | Y | |
| 98 | 130/80 | 79 | 18 | 0.6 | 125 | 5 | 0.7 | 17 | 29 | 7 | 2 | Days | N | |
| ## | 110/66 | 107 | 38 | 1 | 141 | 4.2 | 1.2 | 22 | 16 | 7 | 3 | Days | Y | |
| 56 | 110/70 | 101 | 22 | 0.8 | 140 | 4.9 | 0.7 | 22 | 27 | 6 | 4 | Days | N | |
| 80 | 110/90 | 120 | 26 | 0.8 | 120 | 3.7 | 0.8 | 22 | 22 | 6 | 2 | Days | N | |
| 90 | 130/80 | 96 | 20 | 0.8 | 124 | 4 | 0.6 | 16 | 28 | 6 | 4 | Days | Y | |
| 60 | 110/70 | 89 | 28 | 0.8 | 131 | 4.1 | 1 | 23 | 16 | 6 | 2 | Days | Y | |
| 70 | 100/70 | 120 | 21 | 0.7 | 137 | 4.8 | 0.8 | 17 | 30 | 7 | 4 | Days | N | |
| 64 | 100/70 | 114 | 17 | 0.8 | 135 | 4.1 | 0.7 | 29 | 23 | 7 | 5 | Days | N | |
| 42 | 140/70 | 112 | 32 | 1.2 | 129 | 4 | 0.9 | 45 | 18 | 7 | 5 | Days | Yes | |
| 94 | 100/80 | 145 | 16 | 0.6 | 121 | 4.3 | 0.7 | 18 | 24 | 7 | 3 | Days | N | |
| 60 | 130/80 | 293 | 16 | 0.8 | 143 | 3.5 | 0.8 | 24 | 36 | 7 | 4 | Days | Yes | |
| 90 | 100/70 | 74 | 25 | 0.8 | 125 | 3.8 | 1 | 39 | 38 | 7 | 2 | Days | N | |
| 88 | 100/60 | 76 | 18 | 0.7 | 143 | 4 | 0.8 | 20 | 29 | 7 | 4 | Days | N | |
| ## | 110/70 | 111 | 20 | 0.8 | 138 | V.High | 0.9 | 21 | 24 | 3 | 1 | Day | Yes | |
| 90 | 130/90 | 92 | 37 | 1.4 | 127 | 4.5 | 0.8 | 21 | 25 | 7 | 6 | Days | Yes | |
| 76 | 120/70 | 199 | 34 | 1.2 | 130 | 3.5 | 0.9 | 17 | 18 | 6 | 3 | Days | Yes | |
| 88 | 110/80 | 113 | 27 | 0.9 | 136 | 4 | 0.9 | 25 | 20 | 7 | 5 | Days | yes | |
| 82 | 170/110 | 82 | 18 | 0.6 | 131 | 3.5 | 1 | 28 | ## | 7 | 4 | Days | Yes | |
| 84 | 150/50 | 84 | 22 | 0.8 | 136 | 3.6 | 0.8 | 12 | 18 | 7 | 3 | Days | N | |
| 88 | 120/80 | 120 | 30 | 1.1 | 120 | 3.8 | 0.9 | 16 | 14 | 8 | 3 | Days | N | |
| 80 | 110/70 | 499 | 18 | 0.6 | 126 | 3.6 | 0.9 | 12 | 15 | 4 | 2 | Days | Yes | |
| 55 | 120/70 | 61 | 34 | 1.3 | 130 | 4 | 0.8 | 23 | 37 | 7 | 4 | Days | N | |
| 54 | 110/70 | 160 | 36 | 0.9 | 129 | 5.9 | 0.8 | 29 | 31 | 7 | 4 | Days | yes | |
| 54 | 110/70 | 86 | 30 | 1 | 129 | 5.6 | 0.9 | 36 | 18 | 7 | 3 | Days | Yes | |
| 90 | 140/80 | 120 | 37 | 1.2 | 130 | 3.7 | | 25 | 25 | 7 | 3 | Days | N | |
| 54 | 110/80 | 96 | 25 | 0.7 | 135 | 3.9 | 1 | 33 | 20 | 7 | | | yes | |
| 70 | 120/80 | 132 | 24 | 0.8 | 134 | 3.6 | 0.8 | 34 | 20 | 6 | | | yes | |
| 90 | 110/70 | 111 | 34 | 1 | 128 | 3.6 | 0.8 | 36 | 16 | 6 | | | yes | |

| I.Dopamine | I.kcl | i.insulin | Comments |
|---|---|---|---|
| Yes | Yes | | Idioventricular Rhythym |
| Yes | | | S B |
| | | | S B |
| | Yes | | N |
| | | | N |
| | yes | | S T |
| | | | N |
| | | | N |
| | | | N |
| | | | S T |
| | | | I HB |
| | | | Jn Rhythm, I HB |
| | | | N |
| | | | N |
| | | | N |
| | | | S T |
| | | | N |
| | | | N |
| | | | N |
| Yes | | | Jn RH, II HB Type I block |
| Yes | | Yes | MobitzII |
| Yes | | Yes | I HB, F.S P, Jn.Rhythm, II HB |
| | | | 2:1 AV Block, Rate 45 |
| | yes | | N |
| | yes | | S B |
| Yes | | Yes | Int. 2:1 AV Block |
| | yes | | Jn.Rhythm with Capture Beats |
| | | | I HB |
| | | | I & II Degree Type 1 Block |
| | | | N |
| | | | N |
| | | | CHB,Sinus Arrest |
| | yes | | S B |
| Yes | | yes | II HB Type I &2:1 Variable Block, Int Jn.Rh |
| | | | N |

| | | |
|---|---|---|
| | | S B |
| | Yes | S B |
| | | N |
| | | S P |
| | | S B |
| Yes | | N |
| | | S B |
| | | I.S P |
| Yes | | N |
| | | S P |
| | | N |
| | | S T |
| | | N |
| | | S T |
| | | N |
| | | N |
| Yes | | S P, I HB |
| | | N |
| | | S B |
| | | N |
| | | S B |
| | Yes | I HB |
| | | S B |
| | | I HB |
| | | S B |
| | | I HB |
| Yes | | N |
| | | N |
| Yes | | S B |
| | | N |
| | yes | II Degree Type 1 Block |
| | yes | F.S P, Varying RR int |
| | | N |
| | | Var. II Degree AV Block, F.S P |
| | | S P |
| | | SP,I HB |